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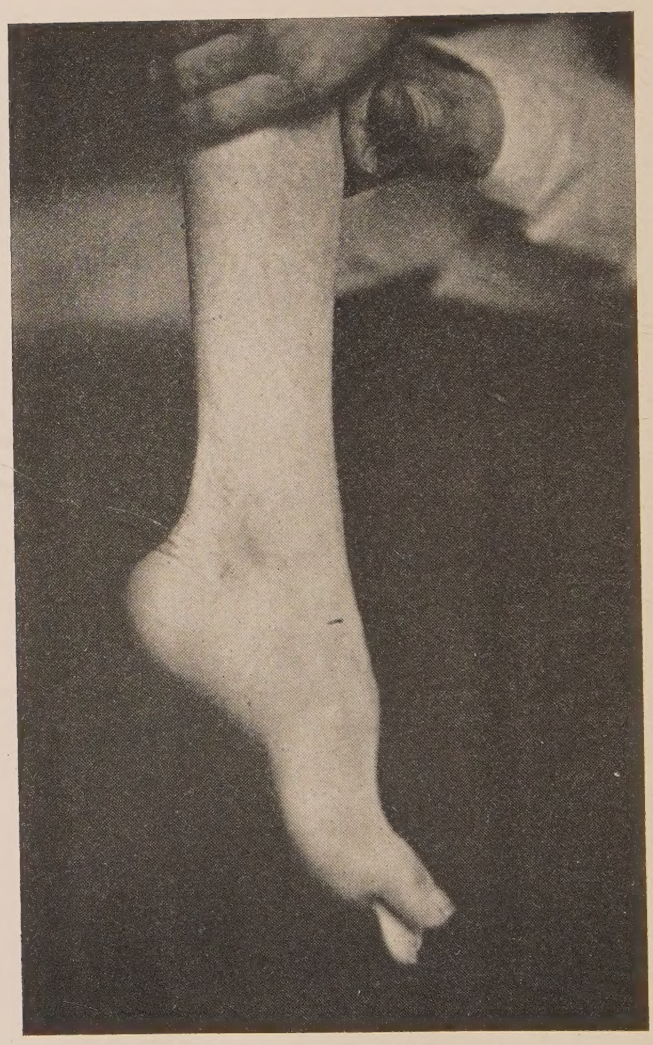

Fig. 1.—Foot of Friedreich's disease.

DIAGNOSTIC SYMPTOMS
IN
NERVOUS DISEASES

BY
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PHILADELPHIA

To My Friend and Chief

M. Allen Starr,

PROFESSOR OF NEUROLOGY, COLUMBIA UNIVERSITY, N. Y.

PREFACE TO THE SECOND EDITION

ORIGINALLY, the demand for this little book came from my students at Columbia University. The first edition filled such a need that a second one proved necessary. This contains new chapters on "Cerebro-Spinal Fluid," "Spinal Localization," and "Vertigo." The outline used at Columbia University for examining a nervous case has also been added. Changes and additions have been made in several other chapters and several pictures have been added.

I again express my thanks and appreciation of the valuable help given me by Dr. Thaddeus H. Ames. I also thank Drs. Leon H. Cornwall and I. J. Levy for the great help they gave me on the chapter on Cerebro-Spinal Fluid.

E. L. H.

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NEW YORK CITY.

PREFACE

My students at Columbia University have asked me for several years to name a book in which they could find the salient points and leading symptoms of the principal nervous diseases without the laborious search involved in consulting the larger text-books.

This book, written to supply such a demand, is intended for the student, intern, and general practitioner, both as a reference and as an aid in diagnosis.

I wish to express my thanks and sincere appreciation for the suggestions and valuable help given me by Dr. Thaddeus H. Ames. He has many times improved, and on one or two occasions added to the manuscript.

The illustrations, nearly all of which are original, were taken from patients on my service in the Central and Neurological Hospital of New York City. For the help in obtaining them I am indebted to Dr. Thomas H. Price.

It has not seemed advisable in a work of this sort to give credit on each page for each reference. A complete list of the books and authorities on which I have drawn is at the end of the volume.

E. L. H.

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DIAGNOSTIC SYMPTOMS IN NERVOUS DISEASES

CHAPTER I

THE EXAMINATION OF A NERVOUS CASE

Proper examination of a nervous case demands skill, intelligence, equipment, and patience. Above all, learn to let the patient tell his own story uninterruptedly. This may take half an hour or more. It, however, affords the examiner an unequaled opportunity to study his patient, to form a tentative diagnosis, and to decide in what particular direction to question him further and more closely. In private practice this method serves the further purpose of greatly increasing the patient's confidence in the physician. At the end of the patient's story first take the history, second examine the patient, third correlate all the facts and then diagnose the case.

History

Begin the history with the statement of the patient's complaints. Then investigate and make a careful study of the patient's antecedents. Go as far back as possible. Give especial care to the nervous and mental heredity. Find out if there is any insanity in the family, any neurotic tendencies, any alcoholism, or epilepsy. Inquire as to the occurrence of cancer, tuberculosis, and syphilis.

The average family will conceal these facts, considering them of minor importance; the student must learn, therefore, that in this regard he cannot be too careful and painstaking.

Next take up the previous history of the patient. Begin with the mother's pregnancy, labor, and delivery. Make a careful study of the infancy, the age at which the patient walked, talked, and teethed; the mode of feeding and the bringing up of the child; the progress of the child at school; then the home life of the individual, together with the bringing up, diseases, and incidents of the next twenty years. Make a careful study into the previous and present sexual life.

Investigate the mental and moral life of the individual; the occurrence of any accident or surgical operation; also the fact as to whether the patient may have been the user of drugs or the sufferer from any toxins. Ascertain the patient's occupation, mode of life, and habits; when he had to give up work, and if because of his illness. Ask definitely whether the patient ever contracted syphilis. Then take up the history of the present trouble. In taking a history of the present complaint pursue a definite order in determining the number and character of the symptoms complained of.

1. As to deformities—determine (*a*) location and distribution, (*b*) whether congenital or acquired.

2. As to pain—determine (*a*) its location and distribution; (*b*) its relation to rest, motion, and sleep; (*c*) its character, whether sharp, dull, shooting, boring, or gnawing; (*d*) its frequency, whether constant, interrupted, or spasmodic; (*e*) what methods of alleviation have been used and with what success.

3. As to motor or paralytic disturbance—determine (*a*) its location and distribution; (*b*) its character; (*c*) its relation to rest, motion, and sleep; (*d*) onset, sudden or gradual; (*e*) degree of incapacity occasioned by symptoms.

4. As to walking—determine (*a*) the character, whether shuffling, uncertain, or high stepping; (*b*) the exact parts of the body affected, whether one or both legs, feet only, or entire side of body; (*c*) whether the patient wears out one or both shoes and in what place; (*d*) whether the difficulty in the gait is stationary or advancing.

5. As to numb sensations or sensory disturbances—determine (*a*) their location and distribution; (*b*) their relation to rest, motion, and sleep; (*c*) their frequency, whether constant, interrupted, or spasmodic; (*d*) their extent, whether slight or severe. Determine whether the sensation is one of needles and pins, of the limb falling asleep, or the lack of the sensation of touch, pain, or temperature, or, finally, whether it is the loss of the sensation as to position of limbs in bed.

6. As to convulsions—determine the nature of the convulsion, with the chronologic sequence of events, the prodromal symptoms, the exact nature of the unconsciousness, the character of the breathing, the position and movements of the patient, the color of the face, the presence or absence of frothing at the mouth, voiding of urine, and uttering of cries; the frequency of the attacks, the severity of the attack, whether increasing or decreasing, and the medication taken.

7. As to disturbances of speech—determine (*a*) whether congenital or acquired, how and when; (*b*) whether constant or spasmodic.

8. As to sphincter control—determine (*a*) character, whether overflow, incontinence, or retention; (*b*) distribution, whether involving bladder or rectum, or both; (*c*) whether of old or recent development; (*d*) whether patient is cognizant of desire to evacuate.

9. As to sleeplessness—determine (*a*) the period of occurrence; (*b*) the nature of sleeplessness, whether continuous or as a result of the patient's waking at frequent intervals; (*c*) behavior of the patient when sleepless, whether quiet, restless, or active; (*d*) causes, whether from pain, anxiety, or digestive.

Examination

When these symptoms have been thoroughly described, proceed to make the *examination*. This examination must be complete and conducted in a systematic way. In no branch of medicine is this more essential than in neurology. The one most important point is to have the patient stripped. Then (1) inspect the patient for the following characteristics: deformities, paralysis, tremors, trophic changes, gait, ataxia, and abnormalities of the eye. A description of the proper examination for these points is given in each one of the following chapters. (2) Make a physical examination of all the viscera by palpation, percussion, and auscultation.

Examine the Patient for Nervous Lesions.—Examine the quality of the skin as determined by palpation. Next take the cranial nerves, testing them in order from the first to the twelfth. Have special bottles for the examination of smell, such as oil of cloves, turpentine, asafetida, etc. Use the ophthalmoscope—both direct and indirect methods. Test the motion of the eyeball up,

down, in, and out. Test for the presence of double images. Have more special bottles for the examination of taste, such as solutions of sugar, salt, and vinegar. Use a camel's-hair brush for examining the sensations of the face. Examine the motion of the eyeball outward, the symmetry of the face, the patient's ability to close the eyelid, to open the mouth, and the direction in which he protrudes the tongue. Examine the hearing; if affected, determine as to the character of the deafness, tubal or nerve. Test the tongue from both a motor and a sensory point of view. Examine the larynx and vocal cords, the muscles of the neck and tongue.

Measure the arms and legs to determine the presence or absence of atrophy. If any paralysis, determine its exact nature and distribution, the strength of flexion and extension. Test the strength of the patient's grip. For this purpose use a dynamometer. Test his ability to raise the arms, to hold them at right angles, outstretched, and above the head. Test his ability to squat, to stoop, and to pick up articles. Test the ability of the patient to stand on one foot alone; on his toes and heels. These latter methods serve to try the various leg muscles. Then test his ability to rise from a prone position.

Examine him for the presence or absence of tremors and fibrillary twitchings. These methods are fully described in the chapter on Tremors.

Inspect for Trophic Change.—To do this begin with the skin and then pursue a systematic method so as to include the joints, muscles, etc. In this connection do not disregard both inspection and palpation of the dorsal artery of the foot. It helps in the diagnosis of trophic disorders of the foot.

Examine the Gait.—The two ways to determine a man's gait are by observing and hearing him walk. A patient is much more apt to walk in a normal and unaffected way if he is not being observed than when he is aware of the fact that he is the object of close inspection. Therefore, contract the habit of observing a patient's manner of walking when he first enters the room. Later have him walk a straight line, either a stripe in the carpet or a crack on the floor. Finally, if it is not too dangerous and difficult, have him walk backward and with eyes shut. Shoes are often so constructed as to modify a gait; therefore, always test a patient's manner of walking with and without shoes. Another important point is to take notice of the manner and location in which a patient wears out his shoes. The second method of examining a gait is by the sense of hearing. Accustom yourself to listen to a patient's steps. The drop-foot flops, the paralytic drags, and the ataxic stamps.

Examine for Ataxia.—This will be found described at length in the chapter on Ataxia. The most important test for ataxia is that known as Romberg's. The examination for ataxia must include not only muscles, but also speech and handwriting.

Examine for Convulsions.—This can readily be done by getting a detailed history. To understand this subject see the chapter on Convulsions.

Examine the Sensory Impulses.—First test the epicritic, the protopathic, and the sense of deep or joint sensibility. Test for touch, pain, and temperature. These methods have all been explained in the chapter on Sensory Disturbances. Next examine, by means of the tuning-fork, the vibration sense; by means of mov-

ing both actively and passively the extremities, the kinesthetic sense; and by means of objects of varying size and shape, the sense of stereognosis.

Examine the Reflexes.—First the superficial, next the deep, and finally the special reflexes. The method employed to test the reflexes varies with each individual and is described at some length in the chapter on Reflexes.

Examine the Eyes as to Shape and Size of Pupils.—Look carefully for any inequality or irregularity of the pupils; for nystagmus and paralyses. Examine the reaction of the pupil to light. To do this place the outstretched hands in front of each eye, alternately admitting light and darkness. Examine the reaction of the pupil to accommodation. To do this have the patient look at a distant object and then at a near one. Test his ability to read and to distinguish colors.

Examine the Speech.—First determine whether the disturbance is one of articulation or in character aphasic. If of articulation, determine which letters are mispronounced and which muscles are involved; if aphasic, determine its character.

Examine the electric reactions as described.

Examine for sphincter control.

Finally make a laboratory examination. This laboratory examination must be very complete. Make a careful routine examination of the urine. Measure the amount of urine passed in twenty-four hours. Make a blood-count. Make a Wassermann test. Make a lumbar puncture. Examine the cerebrospinal fluid for the Wassermann test, the butyric acid test, the globulin content, the cell count, and the colloidal gold reaction. The Wassermann test should be made in varying amounts

of the spinal fluid, that is, in 1 c.c., in 4 c.c., and in 6 c.c. These tests will be treated of in more detail in the chapter on Cerebrospinal Fluid.

Make an *x*-ray Examination.—There are many instances in which the *x*-ray will prove of great assistance both in diagnosis and in prognosis. The student should learn to read *x*-ray plates of the skull and spinal column.

The electric examination is an aid. It serves to differentiate the two different types of paralysis. Again it helps in prognosis by showing the presence or absence of the reaction of degeneration. As a therapeutic measure, electricity is of help.

Make a Mental Examination.—Note the orientation, memory, judgment, perception, train of thought, association, impressibility, reasoning, consciousness, the emotional state, the presence or absence of delusions and hallucinations, etc.

OUTLINES FOR CLINICAL EXAMINATION[1]

A. General Examination.

I. *Inspection.*

1. Face. Note abnormalities of contour and volume, asymmetries and malpositions. Note also mouth and teeth.
2. Neck. Note abnormalities of contour and volume, asymmetries and malpositions.
3. Arms and hands. Note abnormalities of contour and volume, asymmetries and malpositions. Note also whether patient is right handed or left handed.
4. Trunk, including spinal column. Note abnormalities of contour and volume, asymmetries and malpositions.
5. Legs and feet. Note abnormalities of contour and volume, asymmetries and malpositions.

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6. Irregular or unusual attitudes. Inspect patient in standing, sitting, and recumbent positions.
7. Abnormal involuntary movements. Note location, frequency, regularity, amplitude, and how affected by activity, rest, and psychic conditions, such as attention and emotion.
   (*a*) Tremors.
   (*b*) Twitchings.
   (*c*) Choreiform movements.
   (*d*) Athetoses.
   (*e*) Spasms, including tics.
   (*f*) Convulsions.
8. Tegumentary status.
   (*a*) Hair and nails. Note in former abnormal presence, absence and distribution.
   (*b*) Pigmentation, cutaneous and mucosal independent of and dependent upon vascularization.
   (*c*) Sweat secretion.
   (*d*) Vasomotor status.
   (*e*) Œdema.
   (*f*) Unusual markings on skin.
   (*g*) Other surface peculiarities and deformities.
9. General appearance as indicative of mental status.

II. *Mensuration of Corresponding Parts.*

III. *Palpation and Massage.*
1. Consistency of skin.
2. Consistency of muscles.
3. Infiltration of muscles or nerves.
4. Consistency of arteries.
5. Solution in continuity of soft parts.
6. Surface temperature.
7. Glandular status.

IV. *Gastro-Intestinal Status.* Note especially vomiting.

V. *Respiratory Status.*

VI. *Cardiovascular Status.*

1. Pulse.
2. Blood-pressure.
3. Heart.

VII. *Rectal, Vesical, and Sexual Control.* Note especially urgency, frequency, retention, incontinence, and whether latter is diurnal or nocturnal.

B. Special Examination.

I. *Co-ordination.* (*Ataxia, Asynergia.*) Observe motofacient and non-motofacient elements.

1. Equilibratory:
   (*a*) Standing with feet together:
      Eyes open.
      Eyes closed (Romberg's sign).
   (*b*) Standing on one foot:
      Eyes open.
      Eyes closed.
2. Non-equilibratory:
   (*a*) Finger to nose and finger to finger tests.
   (*b*) Pointing and pass pointing.
   (*c*) Heel to knee test.
   (*d*) Passing heel along shin.
   (*e*) Touching with toe an object above foot.
   (*f*) Dysmetria.
   (*g*) Adiadochokinesis. Tested by rapidly repeated simple movements such as pronation and supination.

II. *Gait.* Note phase of support and phase of progression.

1. Walking: slowly, fast; running.
2. Walking with eyes closed.
3. Walking on toes.
4. Walking on heels.
5. Walking backward.
6. Walking sidewise.
7. Fournier's tests:
   (*a*) Abruptly rising from sitting position and walking.
   (*b*) Stopping abruptly on command while walking.
   (*c*) Turning abruptly on command while walking.

8. Walking in a circle, both directions.
9. Walking on a line (Fränkel's test).
10. Stair test.

III. *Performance of Skilled Acts.*

1. Dyspraxia.
2. Apraxia.
3. Speech:
   (*a*) Dysarthria.
   (*b*) Anarthria.
   (*c*) Paraphasia.
   (*d*) Aphasia.
4. Handwriting.

IV. *Reflexes.* In their estimation account should be taken of amplitude and force. Use following terms or signs: "absent" ("O"), "slight" ("1"), "active" ("2"), "very active" ("3"), "with clonus" ("4"), "only elicited on re-enforcement" ("R"), (last two only for deep reflexes). Also indicate equality or inequality, using "=" (equal), ">" (greater than), "<" (less than).

1. Deep (tendon) reflexes.	Right.	Left.
(*a*) Jaw jerk		
(*b*) Pectoral		
(*c*) Biceps		
(*d*) Triceps		
(*e*) Wrist		
(*f*) Radial		
(*g*) Ulnar		
(*h*) Patellar		
(*i*) Suprapatellar		
(*j*) Achilles		
(*k*) Dorsocuboidal		
(*l*) Periosteal		
(*m*) Crossed periosteal		
2. Clonus.		
(*a*) Ankle		
(*b*) Patellar		
(*c*) Wrist		
(*d*) Jaw		

3. Superficial (cutaneous) reflexes.	Right.	Left.
(*a*) Ciliospinal		
(*b*) Supra-umbilical		
(*c*) Suprapubic		
(*d*) Upper lateral abdominal		
(*e*) Lower lateral abdominal		
(*f*) Cremasteric		
(*g*) Plantar and its modifications:		
1. Plantar flexion		
2. Babinski		
3. Chaddock (stimulation below external malleolus)		
4. Oppenheim (inner side tibia)		
4. Gordon (compression of calf)		
6. Schaefer (compression of Achilles)		

V. *Muscle Strength* (*Myasthenic Status*). Note any pareses and paralyses of muscle groups involved in following tests:

1. Rising from sitting position.
2. Rising from recumbent position.
3. Flexion, extension, adduction, abduction, and rotation at various joints.
4. Dynamometer and myasthenometer tests.

VI. *Abnormal Associated Movements.*

1. Pronation sign of Strümpell. Patient sitting. Pronation of forearm when flexed by examiner.
2. Radialis sign of Strümpell. Inability to close fist without dorsoflexion of hand.
3. Thumb sign of Klippel-Weil. Adduction and flexion of thumb when abnormally flexed fingers are extended by examiner.
4. Trunk-thigh sign of Babinski. Patient recumbent. Greater elevation of paretic leg when attempting to rise from a firm surface with arms crossed on chest.
5. Re-enforcement sign of Babinski. Patient sitting with legs hanging free. Extension at knee on hand re-enforcement.
6. Tibialis sign of Strümpell. Patient recumbent. Inability to flex thigh without dorsoflexion of foot.

7. Sign of Marie-Foix. Patient recumbent. Flexion at hip and knee with contraction of tensor vaginæ femoris when foot is strongly dorsoflexed and adducted by examiner.
8. Coughing sign. Patient recumbent. Flexion at hip and extension at knee on coughing.
9. Contralateral associated movements. Seen on paretic side when opposite members are moved by patient.
10. Bilateral identical associated movements.

VII. *Muscle Tone* (*Myotonic Status*) of various muscle groups. Distinguish:
1. Atonic, hypotonic, and hypertonic states.
2. Contraction.
3. Contracture.

VIII. *Myotatic Irritability.*

IX. *Myœdema.*

X. *Electrical Reactions.*
1. Faradic nerve excitability.
2. Faradic muscle excitability.
3. Normal galvanic contraction formula. (CCC>ACC.)
4. Abnormal galvanic contraction formulæ.
5. Erb's myotonic waves.
6. Jolly's myasthenic reaction.
7. Tetany reaction.

XI. *Nerve Hyperexcitability.* (*Tetany.*)
1. Trousseau's sign (compression of arm).
2. Chvostek's sign (percussion of facial nerve).

XII. *General Sensory Examination.*
1. Subjective:
(*a*) Dysesthesias (abnormal sensations):
1. Numbness.
2. Tingling.
3. Prickling.
4. Formication.
5. Sensation of cold.
6. Sensation of heat.

7. Burning.
8. Engorgement and distention.
9. Compression.
10. Sensation of disquiet.
11. Vertigo. External or internal. Direction. Time of occurrence. Exciting causes.
12. Sensations not falling under above heads.

(*b*) Pain:

1. Site. Distribution. (Root zone. Nerve zone.)
2. Character, *e. g.*, constant; fulgurant (lightning); throbbing; dull; sharp.
3. Time of occurrence. Duration.
4. Aggravating circumstances.
5. Alleviating circumstances.

2. Objective. Locate areas of lost, diminished, or increased sensitiveness (nerve or root zones). Note also paresthesias and their type—whether retardation, summation, fusion, allocheiria, or metamorphosis.

(*a*) Tactile sense (thigmesthesia):

1. Loss, diminution, or increase.
2. Patient's localization of area stimulated
3. Touch discrimination (circles of Weber).
4. Paresthesias.

(*b*) Pain sense (algesia). Pin and faradic tests.

1. Loss, diminution, or increase.
2. Paresthesias.

(*c*) Temperature sense (thermesthesia).

1. Loss, diminution, or increase.
2. Paresthesias.

(*d*) Vibratory sense (pallesthesia).

1. Loss, diminution, or increase.
2. Paresthesias.

(*e*) Pressure sense (baresthesia).

1. Loss, diminution, or increase.
2. Paresthesias.

(*f*) Muscle sense (myesthesia) and tendon sense.

1. Loss, diminution, or increase.
2. Paresthesias.

(*g*) Joint sense (arthresthesia).

1. Loss, diminution, or increase.
2. Paresthesias.

(*h*) Posture (statognosis).
1. Loss, diminution, or increase.
2. Paresthesias.
(*i*) Weight (barognosis).
1. Loss, diminution, or increase.
2. Paresthesias.
(*j*) Form (stereognosis).

XIII. *Cranial Nerves.*
1. Olfactory (I).
(*a*) Subjective.
(*b*) Objective.
2. Optic (II).
(*a*) Subjective.
(*b*) Objective.
1. Vision.
2. Perimetry.
3. Eye grounds.
3. Oculomotor (III), trochlear (IV), abducens (VI), and further examination of eyes and eyelids.
(*a*) Ptosis.
(*b*) State of palpebral fissure. Exophthalmus. Enophthalmus.
(*c*) Strabismus.
(*d*) Conjugated eye movements in all directions. Note any limitations of range and dissociations.
(*e*) Nystagmus. Tested with eyes directed forward, laterally, upward, and downward. Note character and direction.
(*f*) Size, shape, and position of each pupil.
(*g*) Reaction of pupils to light, direct and crossed (consensual).
(*h*) Reaction of pupils to convergence.
4. Trigeminal (V).
(*a*) Sensory. (General sensory examination.)
1. Subjective.
(*a*) Dysesthesias.
(*b*) Pain. Site. Character. Time and duration. Circumstances affecting it.

2. Objective. Areas in face and mouth of changed sensitiveness and paresthesias of touch, pain, temperature, etc., as in XII, 2.

(*b*) Motor. Status and movements of muscles of mastication.

5. Facial (VII).

(*a*) Sensory.

1. Subjective (dysgeusia).
2. Objective. Taste anterior two-thirds of tongue. Test each side separately.

(*b*) Voluntary motor. Status and movements of facial muscles, including articulation of labials.

(*b*) Vasomotor and secretory (with sympathetic). Note secretion of tears, sweat and saliva, and vasomotor status of face.

6. Acoustic (VIII).

(*a*) Cochlear portion.

1. Subjective.
2. Objective.
   (*a*) Watch test.
   (*b*) Weber's test.
   (*c*) Rinné's test.

(*b*) Vestibular portion.

1. Caloric tests (Bárány).
2. Pointing test.
3. Rotatory tests.
4. Galvanic test.

7. Glossopharyngeal (IX).

(*a*) Sensory.

1. Subjective (dysgeusia).
2. Objective. Taste posterior third of tongue.

(*b*) Motor (see Vagus).

(*c*) Secretory, salivary.

8. Vagus (X).

(*a*) Position of uvula.

(*b*) Palatal reflexes.

(*c*) Pharyngeal (retching) reflex.

(*d*) Phonation and quality of voice.

(*e*) Position of vocal cords and their movements during phonation.

(*f*) Swallowing.

(*g*) Respiration.
(*h*) Cardiac action.
9. Spinal accessory (XI). Movements of head.
10. Hypoglossal (XII).
(*a*) Trophic status of tongue.
(*b*) Movements of tongue.
(*c*) Articulation.

XIV. *Cranial Morphology.*

XV. *Mental Status* (see Special Blanks).

XVI. *Serology.*
1. Blood. Wassermann reaction.
2. Cerebrospinal fluid.
(*a*) Wassermann reaction.
(*b*) Globulin.
(*c*) Cells.
(*d*) Colloidal gold (Lange).

XVII. *Urine Analysis.*

SUMMARY OF CASE

A. *General Examination.*
I. Inspection.
II. Mensuration of corresponding parts.
III. Palpation and massage.
IV. Gastro-intestinal status.
V. Respiratory status.
VI. Cardiovascular status.
VII. Rectal, vesical, and sexual control.

B. *Special Examination.*
I. Co-ordination.
II. Gait.
III. Performance of skilled acts.
IV. Reflexes.
V. Muscle strength (myasthenic status).
VI. Abnormal associated movements.
VII. Muscle tone (myotonic status).
VIII. Myotatic irritability.

IX. Myo-edema.
X. Electrical reactions.
XI. Nerve hyperexcitability. (Tetany.)
XII. General sensory examination.
XIII. Cranial nerves.
XIV. Cranial morphology.
XV. Mental status.
XVI. Serology.
XVII. Urine analysis.

CARDINAL SYMPTOMS AND SIGNS

1. 9.
2. 10.
3. 11.
4. 12.
5. 13.
6. 14.
7. 15.
8. 16.

SYMPTOM ANALYSIS AND DIAGNOSIS

CHAPTER II

DEFORMITIES

Unusual attitudes occur in many neurological diseases. Some are congenital and are, therefore, in the true sense deformities; others are acquired and are not in the true sense deformities. These conditions occur often; they may be classified as cardinal symptoms of the particular disease in which they are found, and are diagnostic. The student ought to familiarize himself with them and ought to accustom himself to be alert for their presence. For this reason I have devoted a chapter to a short description of some of these irregularities, which I shall call deformities. In this subject it is the faculty of inspection which will bring the richest rewards.

To facilitate references and to systematize the subject I have arranged an outline as follows:

I. Deformities of body as a whole.
   - A. Gigantism.
   - B. Dwarfism.
   - C. Gait.

II. Deformities of the head.
   - A. Size.
   - B. Shape.
   - C. Paralyses.
   - D. Position.

III. Deformities of the spinal column.

IV. Deformities of the extremities.

V. Miscellaneous deformities.

I. Deformities of the Body as a Whole

Gigantism, or *giantism*, means an enlargement of the different parts of the body. It may occur as a freak of nature, but in the majority of cases it means acromegaly.

Fig. 2.—Gigantism.

Dwarfism usually means cretinism.

Gait—for the many and unusual gaits which these patients present, reference is made to the chapter on that subject.

Fig. 3.—Cretinism.

II. Deformities of the Head

A. *Size.*

A head unusually large and bulging occurs with hydrocephalus.

A head unusually small is called microcephalic.

These two conditions are accompanied by impaired or absent mentality.

B. *Shape.*

A marked asymmetry of the head occurs in idiocy.

A flattening of the nasal bridge, together with a prominence of the frontal regions, is diagnostic of hereditary syphilis.

Small, friable teeth, which readily become carious, accompany a neuropathic diathesis.

Teeth which are short and crescentic or notched are known as Hutchinson teeth. They indicate hereditary syphilis.

Pin-point pupils almost invariably indicate one of two things—opium or syphilis.

Unequal pupils may be of congenital origin; they may be an occasional expression of a sympathetic involvement; usually, however, they indicate syphilis, rarely alcohol.

Dilated pupils, large and staring, are strong evidence of atropin or cocain poisoning, of optic nerve atrophy; sometimes they indicate a purely functional condition.

Irregular pupils, that is, pupils in which the contour or outline is jagged, are the result of syphilis.

C. *Paralyses.*

Ptosis of the eyelid means third-nerve palsy.

An eye which cannot be moved to the external canthus, one in which the external rectus is paralyzed, is a symptom of sixth-nerve palsy.

D. *Position.*

The retracted head, rigid and fixed in varying degree, occurs in meningitis.

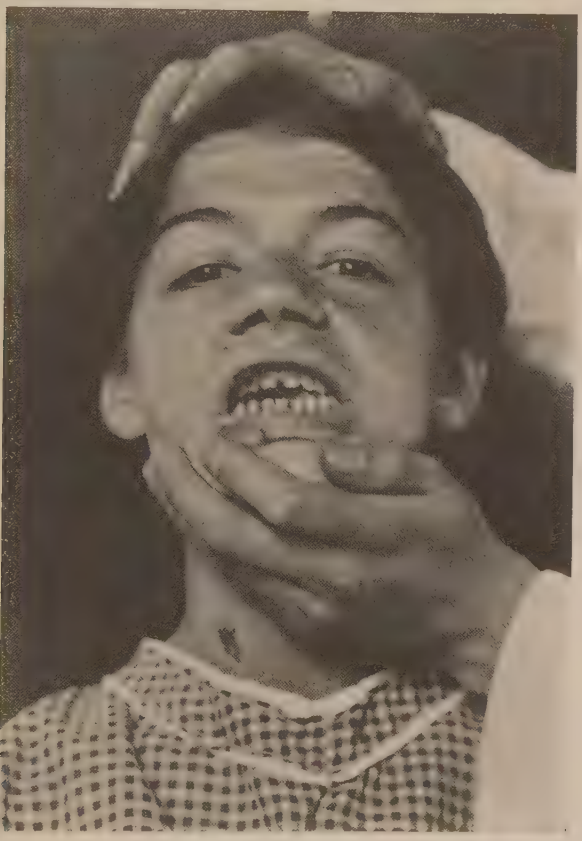


Fig. 4.—Hutchinson teeth.

A head stiff and turned to one side with chin elevated occurs with torticollis.

A head rigid, palsied, and bent forward is characteristic of paralysis agitans.

The head of an old person which trembles or totters indicates senility.

Gaps between the teeth are suggestive of acromegaly.

Bulging eyes, or "pop-eyes," are very suggestive of Graves' disease. They may occur as a result of intracranial pressure. In this instance, however, they would most likely be accompanied by various eye-muscle paralyses.

A retraction of the eyeball, or an enophthalmos, which is the reverse of exophthalmos, is diagnostic of a sympathetic paralysis. The student would do well to connect, with the symptoms of a sympathetic paralysis, the single word "*contracted*," because, in this condition the eyeball is contracted, the palpebral fissure is contracted, and the pupil is contracted.

A "tapir mouth" occurs as a result of some of the forms of progressive muscular atrophy.

Facial atrophy or hemi-facial atrophy occurs in the same condition and in Bell's palsy and poliomyelitis.

Tremor of the tongue generally means alcoholism or paresis.

Tremor of the facial muscles is strongly suggestive of paresis.

A "geographic tongue" means syphilis.

A tongue which is atrophied and paralyzed means bulbar palsy.

A congested or purple face is suggestive of alcoholism, arteriosclerosis, or paresis.

A pallid, wax-like face is suggestive of drug intoxication.

A mask-like face, which is expressionless, rigid, and without muscular tone, slightly flushed, occurs with paralysis agitans.

A face which is expressionless, without muscular tone, flabby, but not rigid, displaying an evident stupidity, is found in paresis.

The face may be as rigid as a mask (the *myopathic facies*) in progressive muscular dystrophy.

The face in Friedreich's ataxia is typical—expressionless.

III. Deformities of the Spinal Column

A curvature of the spinal column occurs in Friedreich's ataxia, syringomyelia, and Pott's disease.

Spina bifida is congenital; it is due to an absence of certain of the vertebral arches.

A knuckle in the vertebral column is the characteristic appearance resulting from spinal dislocation.

Spondylitis deformans is characterized by stiffness and ankylosis of the back, arthritis in other joints, and spinal root symptoms.

IV. Deformities of the Extremities

There are several deformities of the *upper extremities*. All these are important and should be learned.

A single drop-wrist is diagnostic of a peripheral condition, limited to that one particular arm; it means musculospiral or Saturday-night paralysis.

A double or bilateral drop-wrist is symptomatic of a toxic condition. If the arms alone are involved, it is more apt to mean lead paralysis; if the legs as well as the arms are involved, it means alcoholic neuritis.

The paralysis in lead neuritis is of the arms, bilateral.

The paralysis in alcoholic neuritis is quadrilateral.

The paralysis from diphtheria affects successively the throat, eyes, arms, and legs. It is characteristic that

Fig. 5.—Scoliosis (in Friedreich's disease).

one part will improve while another and entirely new one becomes affected.

The paralysis from arsenic is quadrilateral. Starr says that the first symptoms are sensory, and that the pigmented condition of the skin is diagnostic.

The paralysis following grip is apt to take the form of a peripheral neuritis.

A flattened shoulder or an arm which hangs loosely occurring in a child or baby is evidence of the occurrence of an obstetric or birth paralysis. These birth palsies are not uncommon; they are apt to improve as the child grows older. In these cases an *x*-ray picture is valuable; it will disclose the true condition of the joint and the bones.

In the hand occur several deformities.

An ulnar paralysis causes a flexion of the ring and little fingers, though the thumb and other two fingers remain in a normal position. To this deformity has been given the name "preachers'" or "benediction hand." It occurs also in syringomyelia.

The true "claw-hand" is due to paralysis of all the intrinsic muscles of the hand; it is diagnostic of the lower-arm type of paralysis or "Klumpke's paralysis."

The "main de singe," or "monkey-hand," is also due to an atrophy of the thenar muscles; it is the preliminary stage to the "claw-hand."

These last three deformities of the hand occur in lesions of the lower motor neurons, that is, in progressive muscular atrophy, syringomyelia, amyotrophic lateral sclerosis, neuritis, and anterior root lesions. Tapering fingers, the short, square hand, and absent crescents in nails occur in pituitary disease. The skeleton-hand,

or "main de squelette," is one in which the claw-shaped deformity has given place to extension and marked atrophy. It is symptomatic of progressive muscular atrophy.

"Clasp-knife" rigidity, a condition occurring in the legs, is characteristic of primary lateral sclerosis.

A single dropped foot indicates a localized traumatic neuritis. Two dropped feet indicate neuritis, especially alcoholic neuritis, or a complete transverse lesion of the cord.

A club-foot, with a hollow sole, a short and dropped foot, added to a very high instep, occurring in a child or young adult, is the characteristic deformity seen in Friedreich's ataxia.

A retracted heel, one which does not touch the ground, occurs in lesions of the upper neuron type. It is most frequently seen in "Little's disease."

A very red or blue foot is suggestive of erythromelalgia.

V. Miscellaneous Deformities

The wing or angel scapula indicates a paralysis of the serratus magnus and trapezius. It occurs most often in the muscular atrophies.

The dropped scapula is one in which the spine of the scapula forms a more acute angle with the midline than under normal conditions. It indicates paralysis of the rhomboids.

The pot-belly, when seen in children, is strongly suggestive of muscular dystrophy.

Persistent priapism occurring in a paralytic is a symptom of a spinal cord disease.

An attitude of marked rigidity with mask-like face

and accompanied by a coarse tremor is characteristic of paralysis agitans.

A curled-up attitude describes the cerebellar attitude. The patient lies on the side with knees drawn up in a state of flexion. One shoulder is held higher than the other. The side which the patient lies on is generally the side of the lesion. The advanced cases of paralysis agitans and multiple sclerosis are very apt to assume this same attitude. The presence of a tremor, however, will serve to distinguish them.

It may be put down as a general rule that sufferers from locomotor ataxia are thin, while those afflicted with general paresis are fat.

Certain Deformities which are Characteristic of Spinal Cord Diseases

A lateral curvature is frequent with syringomyelia.

A club-foot is characteristic of Friedreich's ataxia.

A dropped foot is found with neuritis, poliomyelitis, and sometimes with myelitis.

A condition of toeing-in is found with the spastic gait; one toe turns in in hemiplegia, the affected foot drops in poliomyelitis.

Conditions in which scoliosis occurs: Friedreich's disease; poliomyelitis; progressive muscular dystrophy; sciatica and syringomyelia.

CHAPTER III

PARALYSIS

THE word paralysis is derived from two Greek words, παρά, meaning beside, and λυείν, to loosen. The medical definition is "abolition of function, whether complete or partial; the loss of power of voluntary motion, with or without that of sensation in any part of the body." *Plegia*, derived from the Greek word meaning "stroke," is another name for paralysis. Palsy is a synonym meaning complete or partial abolition of function. Paresis, on the contrary, although so used, is not a synonym; in this sense its use is incorrect. Paresis means incomplete paralysis, either motor or sensory.

In studying paralysis the student should note three things:

1. The character.
2. The distribution.
3. The degree.

1. **Character.**—By the character is meant the type, whether organic or functional, whether of the upper or lower motor neuron type, or whether peripheral. The large majority of paralyses are organic; a small minority, however, can be classified as functional or hysteric.

2. **Distribution.**—By distribution of paralysis is meant the particular part of the body, the particular muscle, or the particular group of muscles in which the paralysis exists. The importance of this will readily be seen if

the student stops to reflect that there are many different types of paralysis. If, for instance, an entire extremity or one-half of the body is affected, then the paralysis is central in origin; if, on the other hand, one particular group of muscles is affected, then the seat of the paralysis is in the group of cells which supply that particular group of muscles, namely, in the spinal cord; if one individual muscle alone is affected, then the seat of the paralysis is in the nerve which supplies that particular muscle, and is peripheral in origin.

3. **Degree.** By degree of paralysis is meant the extent to which the loss of function extends, that is, whether partial or complete. The affected muscle may be involved to a slight extent, so that a little effort may bring forth the paralysis; again, the weakness may be more marked, so that the muscle itself may be unable to perform any duty.

To understand paralysis it is necessary to form a clear conception not only of the meaning of the word, but also the meaning of the term. Paralysis is a symptom—a symptom that may occur in many affections of the nervous system. It may occur in diseases of the brain, of the spinal cord, or of the peripheral system. It may occur as a symptom of functional diseases, for there is a well-recognized group of diseases known as the hysteric paralyses. Again, paralysis is a term used to describe a particular group of symptoms or a particular disease, as Landry's paralysis.

Classification.—The best way is to divide all paralyses into two great classes: *organic* and *functional*. Then subdivide the organic group into three classes: the *upper motor neuron type, the lower motor neuron type, the*

peripheral type. This division is not arbitrary, but depends upon whether the lesion is situated in the upper or the lower part of the motor tract or in the periphery.

The motor tract extends from the cortex of the brain through the pons and medulla down along the pyramidal tracts, through the spinal cord and its anterior horns to the motor end-plates in the muscles. The lesions of the two kinds of paralysis occur in this tract. The lesions of the first form occur in that part of the tract which extends from the cortex to the cells in the anterior horns of the cord at the termination of the pyramidal fibers. This type of paralysis is called the *upper motor neuron type*, or the *corticospinal*. The lesion of the second form occurs in that part of the tract which extends from the cells in the anterior horn to the muscle end-plates. This type of paralysis is called the *lower motor neuron type*, or the *spinomuscular*.

These two types of paralysis are wholly different, one from the other; the degeneration of the one does not spread into the other; the symptoms of the one are wholly different from those of the other. In each there is a degeneration with this distinction: In the first, the upper motor neuron type, the degeneration does not extend downward; it is rather a degeneration which is universal or complete. At once it affects the entire pyramidal tract. In the second, or the lower motor neuron type, the degeneration does extend downward from the cell in the anterior horn.

The essential point for the student to keep in mind is that any form of motor paralysis must occur in one of these three types, and that, before he can proceed at all, he must be able to locate the lesion.

Many names, such as the rigid and the flaccid, the non-atrophic and the atrophic, have been suggested as suitable for these two types. It will, however, be seen that these names are derived wholly from the symptoms. They are misleading and incomplete. The most comprehensive division is the one which is here adopted—the upper motor neuron and the lower motor neuron types.

Symptoms of Paralysis

The symptoms which these two types of paralysis present are wholly different, and have different causes. There follows a table of the principal symptoms of each:

Symptoms of the Upper	Symptoms of the Lower
1. Rigidity.	1. Flaccidity.
2. Reflexes active.	2. Reflexes absent.
3. No atrophy.	3. Atrophy
4. Electric reactions normal.	4. Reaction of degeneration.

To go a little more into detail, the following table from Stewart shows additional symptoms of each condition:

Upper Neuron	Lower Neuron
1. Diffused muscle groups affected, never individual muscles.	1. Individual muscles may be affected.
2. Spasticity and hypospasticity of paralyzed muscles.	2. Flaccidity and atonicity of paralyzed muscles.
3. May have superadded associated movements on attempted voluntary movement.	3. No associated movements.
4. No muscular atrophy, except from disuse.	4. Atrophy of paralyzed muscles.
5. Electric reactions normal.	5. Reaction of degeneration.
6. Deep reflexes in paralyzed limbs present and usually increased.	6. Deep reflexes of paralyzed muscles diminished or absent.
7. If foot affected, plantar reflex, extensor in type.	7. Plantar reflex, if present, is normal flexor type (unless flexors of toes are themselves paralyzed).

It is not necessary to remember all of these seven differences. The smaller table will answer every purpose. Base the difference upon the location of the pathologic lesion in the motor tract. Divide the two types into upper motor neuron and lower motor neuron. It is the level of the lesion, therefore, which will determine

Fig. 6.—Atrophy of hands occurring in amyotrophic lateral sclerosis.

clinically the physical signs and symptoms. In the case of the lower type, loss of reflex is the best guide; in the case of the upper, exaggeration of reflex.

A combination of the two types may occur. When this happens, the symptoms at the level of the lesion will be those of the upper type, while below the level the symptoms will be those of the lower type. This is due to the

fact that at the level of the lesion there is destruction of the anterior horns and roots; while below the level there is interruption of the pyramidal tracts, as a result of this very destruction of the anterior horns and roots.

I. List of Diseases which Occur Following a Lesion of the Upper Neuron Type

1. Apoplexy.
2. All forms of cerebral diseases.
3. Syphilitic paraplegia.
4. Transverse lesions of the cord.

The student, having settled to his satisfaction, from the foregoing rules, to which of the two types the particular paralysis is confined, has now to determine the particular level in that type.

The Plegias

In this upper type of paralysis there occur certain unusual terms which are distinctive and instructive. The student ought to familiarize himself with their meanings and their significance. As they all have the same termination, I have called them the plegias. The plegias are:

1. Monoplegia.
2. Hemiplegia.
3. Diplegia.
4. Triplegia.
5. Paraplegia.

Monoplegia means paralysis of one extremity only. It is usually cortical in origin for two reasons: First, a cortical lesion which involves more than one extremity must, of necessity, be very extensive; therefore, the pa-

ralyses of single extremities, the cause of which are small lesions, are usually cortical. Second, it is unusual to find a subcortical lesion which occurs in any place other than where the motor tract is united and compact.

Hemiplegia, taken in its literal sense, means paralysis of one half. In neurology the term is used to designate a paralysis of one-half of the body.

The cause of hemiplegia is a lesion in the motor tract. The fibers of this tract decussate in the neighborhood of the medulla, so that those arising in the right hemisphere pass down the left side. Therefore, a lesion on one side of the brain will give rise to a paralysis on the opposite side of the body. The paralyzed side is the side opposite to the lesion. It is a curious fact that in the paralysis which follows apoplexy—for hemiplegia is one of the results of apoplexy—the upper third of the face is effected to a much less degree than is the lower two-thirds. Several reasons have been given to account for this; the one which is most probable, and the one which receives the most general acceptance, is that the upper third of the face receives its innervation from both sides of the brain, and that, therefore, when the fibers from one side are cut there still remain those from the other. This sign assumes considerable importance when one is called to make a differential diagnosis between hemiplegia and Bell's palsy.

Hemiplegia may be acute or chronic, may develop slowly, or may be the symptom of an apoplectic stroke; it may progress, or it may tend to get well; it may even be transitory, as is often the case in general paresis and syphilitic conditions.

It will readily be seen that in hemiplegia the extent

of the paralysis is so considerable that one of two conditions must occur: either the lesion itself must be very extensive, or the part which it affects must be very compact. As a matter of fact, the lesion of hemiplegia is in the motor tract at a point where all the fibers are united, so that a lesion of the size of a pea would interfere with the expression of impulses down the fibers which involve the entire side—the leg, arm, and face. If the lesion extends posteriorly from the motor tract, there will be sensory symptoms.

Hemiplegia may follow:

1. Traumatic lesions of the brain and cord.

2. Meningeal lesions due to hemorrhage, inflammation, syphilis, and tuberculosis.

3. Cerebral lesions due to hemorrhage, softening, tumors, abscesses, and sclerosis.

4. Cerebrospinal lesions of tabes, multiple sclerosis, and general paralysis of the insane.

It may be due originally to:

1. Intoxication by uremia, diabetes, alcohol, lead, mercury, and some carbon compounds.

2. Infections from pneumonia, malaria, typhoid fever, puerperal fever, eruptive fevers, diphtheria, influenza, syphilis, and tuberculosis.

3. It marks some cases of chorea and hysteria.

Of the entire half of the body affected, the arm is usually the part most paralyzed, the leg next, and the face least of all. The speech at first is always involved. The permanent impairment of the speech, however, depends upon whether the lesion is situated on the left side or not. If it is on the left side, the speech

center is involved, and the patient becomes aphasic. If, on the other hand, the lesion is on the right side, there is no involvement of the speech center. However, there is paralysis of the muscles of the tongue and throat, regardless of which side is affected, and this of necessity results in a more or less complete disturbance of speech. This disturbance of speech is most distressing to the friends, and one which requires explanation.

A good practical point to remember is that by observing the relationship of the paralyzed side to the speech center it is possible to make a definite and immediate prognosis as to the recovery of speech. The only exception to the rule is in the case of a left-handed person. Such persons have the speech center on the right.

The type of paralysis which occurs in hemiplegia is, of course, that of the upper motor neuron type. There are, therefore, exaggerated reflexes, rigidity, and an absence of any electric changes. After the disease has lasted some time there will develop slight atrophy which is due to disuse.

The gait of hemiplegia is characteristic, and is described elsewhere.

A striking characteristic that the patient presents is one of always pulling the affected arm with the uninjured one.

The most important sequelæ which follow hemiplegia are tremors and athetoid movements.

Varieties.—There are several types of hemiplegia which merit a word and need elucidation.

Crossed or *alternating hemiplegia* is the name applied to that form in which the paralysis is distributed to the face of one side and the leg, arm, and trunk of the opposite side. It is the result of a lesion in the pons.

Double hemiplegia is also known as *diplegia*. It is of rare occurrence. Little's disease is a synonym.

Infantile hemiplegia is simply the term applied to a hemiplegia which occurs in children. It differs but little from the ordinary form. The main differences are in the nature of the underlying lesion and in the time of onset.

Hysteric hemiplegia is not uncommon. This is a one-sided paralysis. It differs from the organic in many respects—in the gait, the presence of stigmata of degeneration, and the areas of anesthesia.

Diplegia is double hemiplegia.

Triplegia is paralysis of three extremities. It occurs as a combination of monoplegia and hemiplegia, or as a combination of paraplegia and monoplegia; it might occur in poliomyelitis.

Paraplegia is paralysis of the two lower extremities, or the two upper. The most common form in which it occurs is spastic paraplegia.

Spastic Paraplegia.—The words spastic paraplegia have probably confused and misled more students than any other term in medicine. The first thing to learn about the term spastic paraplegia is that it does not mean a disease. The next thing to learn is the meaning of the words. *Spastic* means stiff; *paraplegia* means paralysis of the two lower extremities. The term, therefore, applies not to a disease, but to a condition. A diagnosis of spastic paraplegia is no diagnosis at all. The information that such a diagnosis conveys is simply that the patient is afflicted with stiffness of the lower extremities. This condition has its cause, and it is this cause which would be the correct diagnosis.

Affections of the Nervous System Causing Spastic Paraplegia

A. Spastic paraplegia secondary to other spinal diseases.	Compression myelitis from— Vertebral caries. Vertebral tumor. Tumor of the spinal cord and its membranes. Spinal hydatid. Spinal compression from aneurysm. Fracture dislocation. Gunshot and other wounds of the cord. Acute transverse myelitis and disseminated myelitis. Hematomyelia and meningeal hemorrhage. Spinal meningitis and pachymeningitis. Syphilitic myelitis, meningomyelitis.
B. Spastic paraplegia usually associated with other symptoms.	Amyotrophic lateral sclerosis. Disseminated sclerosis. Syringomyelia. Ataxic paraplegia. Combined posterolateral degeneration and sclerosis associated with anemia and various toxic conditions. Erb's syphilitic spinal paralysis. Occasionally general paralysis of the insane. Cerebral diplegia (Little's disease).
C. Primary spastic paraplegia.	Primary lateral sclerosis and hereditary spastic paraplegia.

The preceding table on p. 56, taken from Williamson, is the most complete of its kind, and gives a list of the diseases in which spastic paraplegia occurs:

The condition known as spastic paraplegia requires three distinct characteristics to make it complete:

1. Loss of power in both legs.
2. Rigidity of both legs.
3. Exaggeration of the deep reflexes.

II. List of Diseases which Occur Following a Lesion of the Lower Neuron Type

1. Poliomyelitis.
2. Amyotrophic lateral sclerosis.
3. Myelitis (localized or extensive).
4. Syringomyelia.
5. Tumors and hemorrhages within the cord.
6. Softening of the cord, due to embolus or thrombus.
7. Neuritis.

III. List of Diseases in which the Combined Lesion Occurs

1. Myelitis.
2. Myelomalacia.

It will be of inestimable aid to the student if he will accustom himself to group in his mind the several symptoms that go to make up these two types of paralysis. Thus, he should remember that rigidity and exaggerated reflexes constitute the only unusual signs of the first type, because in that type there is neither muscular nor electric change. Therefore, if he will fix in his mind in connection with the symptoms of the upper type the single letter R he will readily be able to remember the two principal symptoms which denote that type. R stands for *rigidity* and *reflexes* plus.

If, in connection with the lower type, he will fix in his mind the fact that in that type the symptoms are exactly the reverse of those in the upper type, he can readily reason them out.

IV. Peripheral Paralyses

The principal train of symptoms which occur in the peripheral paralyses are as follows:

1. The loss of power.
2. The characteristic deformity.
3. The atrophy of the muscles.
4. Congestion, edema, and sweating.
5. Twitchings (if damage is slight or if irritated).
6. Pain.
7. Sensory symptoms.

The motor symptoms depend on whether the nerve is a motor nerve; the sensory, on whether it is a sensory nerve. In a few instances a nerve may contain both fibers.

The most important of all these symptoms to the student is the characteristic deformity.

Below is a list of the important *arm paralyses*, with the characteristic deformity of each:

1. Obstetric paralysis or birth palsy.
   Flail-like arm with flat shoulder.
2. Circumflex paralysis.
   Atrophy of deltoid; triangular area of anesthesia.
3. Musculospiral paralysis.
   Dropped wrist.
4. Ulnar paralysis.
   Benediction hand.

5. Median paralysis.

No characteristic deformity, but hand supinated and inability to flex fingers.

List of *leg paralyses:*

1. Sciatic.

The dragged leg.

Fig. 7.—Dropped feet.

2. Peroneal.

The pigeon-toe foot.

3. Anterotibial.

The dropped foot.

4. Internal plantar-nerve paralysis causes paralysis of the toe and deformities of the big toe.
Hammer-toe.

To this list may be added contracture of the tendo Achillis, horse-foot. Of these paralyses the obstetric and the circumflex are the most obstinate, the musculospiral the one most amenable, to treatment.

Miscellaneous Paralyses

There follows a list of miscellaneous paralyses. Some derive their names from men who first described them; some, from the condition that produces them; still others from the clinical symptoms they present, and finally still others from the occupation causing them. Each paralysis is placed in a paragraph by itself, containing the correct medical name of the condition, with a few words describing its cause, principal symptoms, and course.

Saturday-night Paralysis.—The correct name for this is musculospiral paralysis. It arises from the drunkard's falling asleep upon his upper arm and remaining so for several hours. The cause, then, is twofold—pressure and pressure exerted upon a nerve poisoned by alcohol. The deformity is dropped wrist. In the vast majority of cases it occurs in the lower grade of humanity following a Saturday-night debauch.

Bell's Palsy.—Bell's palsy is another name for facial paralysis. The name is derived from Sir Charles Bell, who first described it. It is a paralysis of the seventh cranial nerve and results in a more or less complete paralysis of one side of the face. The cheek is flattened; the muscles are drawn to the opposite side; the patient cannot whistle and is unable to control the lips of the

affected side. The paralysis involves all the muscles of the side of the face. The eye stares. The characteristic feature, however, is the inability on the part of the patient to close the affected eyelid. In these cases the student ought to practise a close observation of the eyelid. If the patient is able to shut the affected lid, the lesion is in the motor tract and not in the course of the seventh nerve. If he is unable to do so, the condition is Bell's palsy. Along with this inability to close the lid there may be an involvement of taste, saliva, and hearing, according as to what particular part of the nerve is involved. The location of the lesion may be in one of four places:

1. In the pons.

2. Between the pons and the geniculate ganglion, that is, above the geniculate ganglion.

3. Between the geniculate ganglion and the emergence of the nerve from the stylomastoid foramen, that is, below the geniculate ganglion.

4. On the face after the nerve emerges from the stylomastoid foramen, that is, below both the geniculate ganglion and the stylomastoid foramen.

In 1 the lesion is in the pons; the symptoms are those of facial paralysis, only there is no involvement of either taste, saliva, or hearing.

In 2, where the lesion is above the geniculate ganglion, the symptoms are those of facial paralysis, with usually an involvement of hearing. If there is no paralysis of the auditory nerve, stapedius paralysis will result and hyperacusis.

In 3, where the lesion is between the geniculate ganglion and the stylomastoid foramen and within the Fallo-

pian aqueduct, the symptoms are those of facial paralysis, plus loss of taste in the anterior two-thirds of the tongue. If the nerve to the stapedius is involved there may also be hyperacusis. If the geniculate ganglion itself is inflamed, there will be added herpes on the ear. The important points, then, are the presence or absence of taste in the anterior two-thirds of the tongue and the involvement of hearing.

In 4, where the lesion is below the stylomastoid foramen, the symptoms are those of facial paralysis, only there is no involvement of either taste, saliva, or hearing.

Landry's paralysis, or acute ascending paralysis, is the name given to a motor paralysis, which most resembles that of the lower motor neuron type. It begins in the lower extremities, extends upward until some vital organ or center is involved, and then ends fatally. The two principal characteristics of this condition are its total disability and the great rapidity of its progress. There is no pain, there may be a slight numbness. The course of Landry's paralysis is a few days.

Divers' Paralysis.—This is another name for caisson disease, a condition to which the laity, particularly the victims, have applied the term of "the bends." It is a form of myelitis, comes on suddenly, lasts for a short time, and, as a rule, leaves the patient uninjured.

The condition is common in divers and all workers under compressed air. It is due to the sudden removal from the spinal cord of an extraordinary pressure.

Paralysis appears only when the surface is reached, and requires a pressure of two atmospheres.

Infantile Paralysis.—This is another name for polio-

myelitis, an acute, infectious disease of the lower motor neuron type. The term is not a good one, as one is apt to confuse it with the cerebral palsies of childhood.

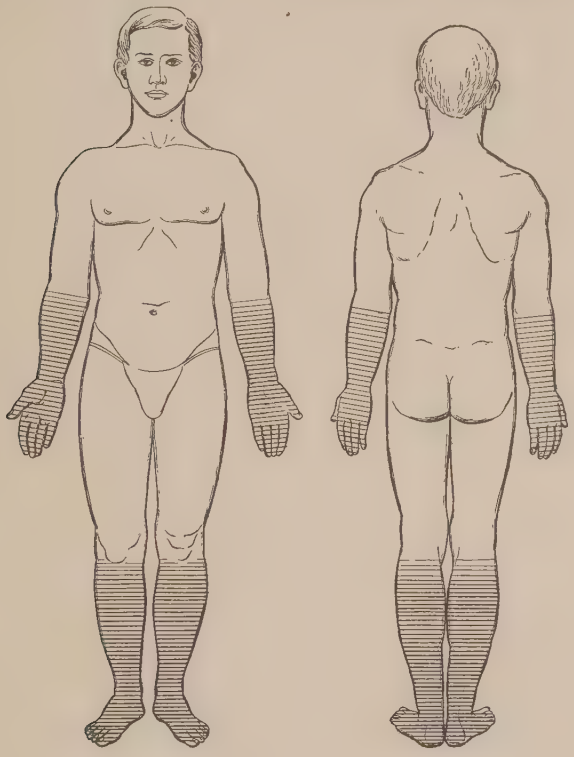


Fig. 8. Fig. 9.

Figs. 8, 9.—Area of pain in multiple neuritis.

Stocking and Glove Paralysis.—This is a term applied to the sensory disturbance which occurs in the course of peripheral neuritis. The sensory disturbances are supposed to extend as far up the extremities as do long gloves and stockings.

Bulbar Paralysis.—A synonym for glosso-labio-laryngeal paralysis. It is a progressive atrophy of the motor neurones of the ninth, tenth, eleventh, twelfth, seventh, the motor part of the fifth and sometimes of the sixth and third cranial nerves. The patient has difficulty in swallowing, coughing, and talking.

Diphtheric paralysis is a term applied to the paralysis which follows diphtheria. The symptoms are paralysis of the soft palate, of the eye muscles, and of the legs.

Erb's paralysis is a name applied to the upper-arm type. The lesion involves the central parts and upper cords of the plexus.

Shaking palsy is another name applied to paralysis agitans, a disease in which the characteristic symptoms are tremor, rigidity, and festination.

Sympathetic paralysis is a term applied to an involvement of the cervical sympathetic nerve. The symptoms are slight retraction of the eyeball, narrowing of the palpebral fissure, slight contraction of the pupil, slight drooping of the outer angle of the eye, and a dryness of the skin on the affected side.

Bernhardt's paralysis is a peripheral neuritis of the external cutaneous nerve of the thigh. The symptoms are pain and diminished sensibility of the skin.

Klumpke's paralysis is the name applied to a lower-arm type of paralysis. It was first described by Flaubert in 1827. The cause is an overstretching of the brachial plexus. The characteristic deformity is the claw-hand.

Crutch palsy is a name applied to a pressure neuritis of the nerve-trunks in the axilla. It is the result, as the name indicates, of prolonged and improper use of a crutch.

Brown-Sequard's paralysis is a condition, the result of an affection of one lateral half of the cord. The symptoms differ on the two sides.

Symptoms on Side of Lesion	Symptoms on Opposite Side
1. Paralysis with rigidity.	1. No paralysis.
2. Reflexes plus.	2. Reflexes slightly increased.
3. Vasomotor paresis.	3. Loss of touch, temperature, and pain up to a line passing about the body one inch lower than the zone of anesthesia.
4. Muscular sense diminished.	
5. Touch, temperature, and pain hypersensitive.	
6. Narrow zone of anesthesia to touch, temperature, and pain.	

Duchenne's Paralysis.—This is another name for the obstetric paralysis.

Lead paralysis is the name given to one of the forms of multiple neuritis caused by lead—a paralysis of the extensors in which the supinator longus escapes. Characteristic—dropped wrist.

Arsenical paralysis is one of the forms of multiple neuritis caused by arsenic. The distinctive symptoms are sensory and gastro-intestinal.

Gas paralysis is one of the forms of paralysis due to poisoning by carbon monoxid.

Patheticus paralysis is another name for fourth-nerve paralysis.

Third-nerve Paralysis.—(See chapter on Eye.)

Abducens Paralysis.—(See chapter on Eye.)

Compression Myelitis.—This is a condition resulting from changes in the spinal cord, which, in turn, are caused by a reduction of the space in the spinal canal. The most common causes of a compression myelitis are as follows:

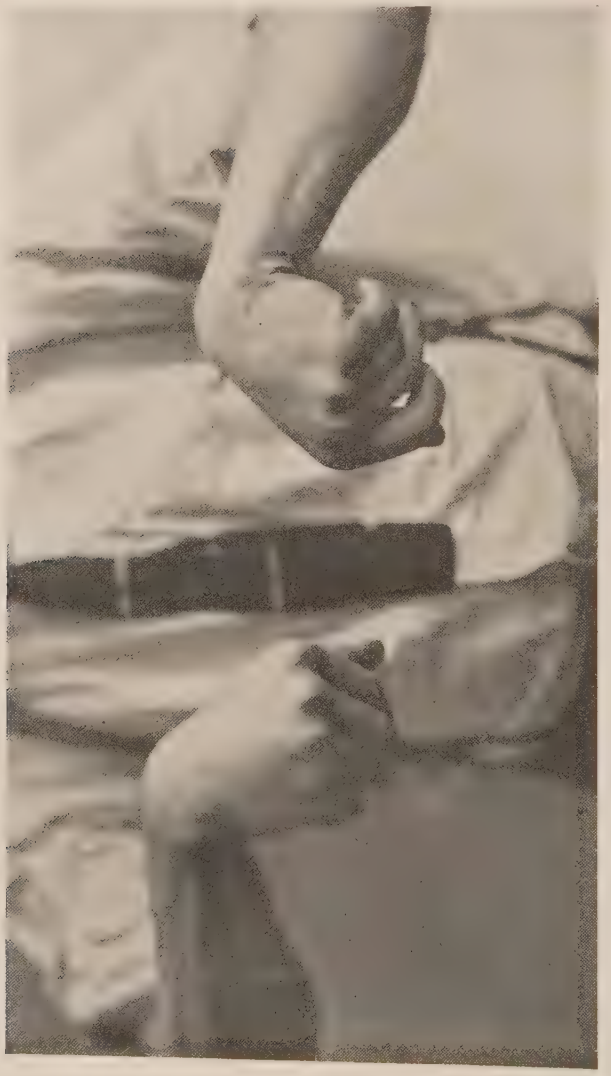


Fig. 10.—Dropped wrists in lead palsy.

1. Caries of vertebræ (tubercular, very rarely syphilitic).
2. Tumors of the vertebræ.
3. Meningeal and extrameningeal tumors.
4. Spinal hydatid cysts.
5. Aneurysms eroding the vertebræ and compressing the cord.
6. Chronic hypertrophic pachymeningitis.
7. Fractures and dislocations of the vertebræ.

CHAPTER IV

TREMORS

Tremor means trembling. The word is derived from the Latin *tremere*, meaning to tremble. In medical nomenclature tremor is used to designate the abnormal, rapid contractions which occur in a muscle group, not, however, those contractions which pass from one group to another. All muscles in their normal condition are kept in a state of slight tension by impulses, which pass down in a regular, rhythmic manner. When these normal impulses are interfered with, either by increasing or interrupting some of them, there results a tremor. The manner in which these impulses are interfered with enables us to classify tremors in an intelligible, systematic manner. Thus, if these impulses are increased both in number and force, the tremor is fine; if these impulses are diminished both in number and in force, the tremor is coarse.

Many ways to classify tremors have been suggested and adopted. Each text-book has a different method. Each instructor has tried his own way. Tremors have been divided into constant and inconstant, into regular and irregular, into intention and non-intention, into fast and slow. Each classification presents a difficulty of its own; each is confusing to the student; none is complete, and not one enables him to place any tremor in one class or the other. The simplest, the most rational, the easiest, way for the student is to divide all tremors into two classes: coarse and fine. The advantage of this

classification is that any and all tremors can be placed under one of the two headings; thus, the intention tremor, though in a class apart, is yet coarse; the alcoholic tremor, though toxic, is yet fine.

The study and observation of tremors will materially help the student and aid his efforts in reaching a diagnosis. In studying tremors the one important point to remember is to cultivate the powers of observation. I make this emphatic because so many tremors are aggravated by outside influences. It is of great value to the student to learn the habit of observing a patient as he enters the room, of carefully noting his gait and bearing, of noticing the presence or absence of a tremor.

In order to make a successful, thorough examination of a tremor the student should note the following facts:

1. The distribution.
2. The rate and rhythm.
3. The effect of rest.
4. The effect of voluntary movement.
    (*a*) Raising a glass of water.
    (*b*) Writing.
    (*c*) Walking.
5. The effect of external influences.
6. Cardinal signs of the accompanying diseases.

It is well to consider a little more in detail these six methods.

1. The **distribution** of the tremor means what particular part of the body is trembling, and just what particular group of muscles is trembling. Not only is this of service in differential diagnosis from the point of view of eliminating certain other portions of the body, but it also affords information as to the particular tremor.

Particular tremors affect particular parts of the body. Thus, a tremor of the head is almost invariably senile; a tremor of the facial muscles almost invariably toxic. It is, therefore, of great importance thoroughly to go into the matter of distribution. This cannot be done unless the examination is systematic. It is best, therefore, in examining for the distribution of a tremor, to begin at the head, completely to examine the muscles of the face, then the arms, and, finally, the trunk and legs. The best way to examine the facial muscles is to have the patient clinch the teeth, pushing the lips back as far as he possibly can, in imitation of the grimace of grinning. The way to examine the hands is to have the patient extend them with fingers spread out.

2. Under the head of **rhythm of the tremor** are included several things, such as the rapidity of oscillation, the regularity, and the amplitude. The rapidity or slowness of a tremor is one of the important differential diagnostic points. In a fast tremor the oscillations are from 10 to 20 per second; in a slow tremor they are below 5. A tremor that is rapid has, as a rule, small excursions, whereas one that is slow presents more extended excursions. The regularity of the oscillations is really covered under the other headings.

3. **The Effect of Rest.**—To determine whether the tremor occurs during rest as well as during active motion is equally important. Here the same rule applies. Certain tremors occur during one condition, certain during another. To decide the question, the patient ought first to sit down, then to lie down wholly relaxed. Bed is the best place, the head resting upon a soft pillow. Inspection in this position is generally sufficient. If

inspection fails to disclose the presence of any tremor, resort should be had to palpation. The examiner should place his hand upon the patient's head, shoulders, and extremities. The finest, most invisible trembling, under these conditions, becomes apparent.

4. The way to determine the **effect of voluntary movements** is: first, for the examiner to raise the limb and hold it extended; next to have the patient raise and extend the limb; and, finally, to have the patient perform more complicated movements, such as following the examiner's finger or picking up articles. The classic way of making this test is to have the patient hold extended a glass brimful of water, and then raise it to the lips. If the glass is full, the slightest, gentlest oscillation will alter the level of the fluid.

To examine the arms for tremor, have the patient draw a straight line, touch the extended arms of the examiner, hold the hands outstretched, and write.

Much the same methods can be used to examine the legs. First the patient should lie down, then sit, then extend the leg, and, finally, walk.

5. **The Effect of External Influences.**—Note the effect upon the tremor of attention, self-consciousness, and mental influence. These help to determine the condition as organic or functional. Ability to use the extremities is of value in illustrating whether the tremor is affected by external influences, and whether or not it belongs to the class known as "intention tremor." A display of the handwriting and the gait should give valuable information. Thus, the handwritings of the senile and paretic are both characteristic. The gait of the sufferer from paralysis agitans is distinctive.

Division of Tremors

1. Coarse.
2. Fine.

This distinction, as I have said, is based on two characteristics, the amplitude of the oscillation and the rate of trembling. A *coarse tremor* is one in which the amplitude of the excursion is considerable, the speed slow. A *fine tremor* is just the reverse. A coarse tremor will have few movements, as compared with a fine: 5 to the second, as compared with 10 to 20 in the fine. A coarse tremor is one in which the excursion is large, jerky, slower, and more evident to the eye. A coarse tremor can be illustrated by this diagram:

Fig. 11.—Coarse tremor.

and a fine tremor by this one:

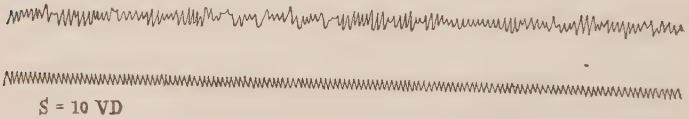


Fig. 12.—Fine vibratory tremor.

The Various Tremors

The following list of tremors will enable the student to understand them thoroughly:

The Intention Tremor.—The distribution of the in-

tention tremor is not limited to the hands or arms, but extends to the muscles about the scapula and neck. It may even involve the legs or any other muscles. Its most common distribution is in the arms.

The intention tremor is coarse, unusually jerky, and possesses considerable amplitude of excursion. Its name is derived from a distinctive trait. This trait is that the tremor is present only when the patient performs a

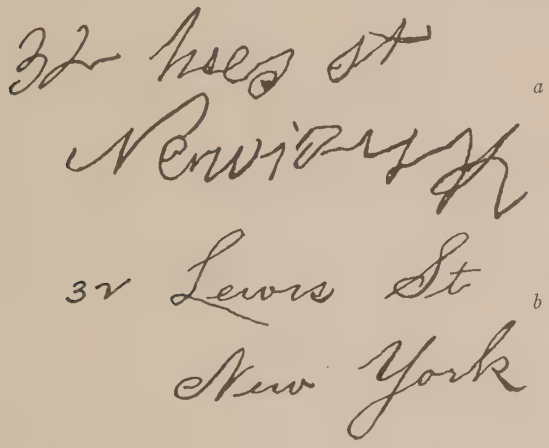


Fig. 13.—*a*, Handwriting of a patient suffering from multiple sclerosis. *b*, Normal handwriting of the same matter.

voluntary action; in short, does something. The hands at rest evince no tremor. So soon as the patient attempts anything, to perform any act, the tremor appears. The best exhibition of the intention tremor is seen in the patient's attempt to drink a glass of water. As he sits at the table, with arms hanging loosely by the side, or resting, possibly on the table, no trembling is apparent. At the moment, however, that he raises his hand and

carries the glass to the mouth, there appears in both arms and hands a tremor coarse and jerky in character, slow in rate, and displaying considerable amplitude of excursion. These movements augment in both size and frequency the more prolonged is the effort which originates them. If the intention tremor is at all pronounced, the contents of the glass will be spilled.

The handwriting is involved early. Wilkinson describes it as being jerky, especially at the end of a word or sentence. The affection of straight lines is not so characteristic as is the occurrence of a sudden, jerky irregularity. The patient attempting to draw a straight line resorts to a series of disconnected blots and jerky, short lines. The walk of a patient suffering from an intention tremor of the legs displays much the same characteristic. It is unsteady and irregular. He stands well, but so soon as the legs begin to move there develops irregularity.

The effect of external and emotional influences on the intention tremor is to increase it. This tremor is one of the cardinal signs of multiple sclerosis. The other three are nystagmus, scanning speech, and exaggerated knee-jerks. An excellent diagrammatic representation of the intention tremor is the one given by Charcot:

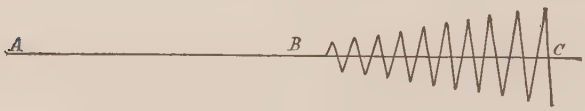


Fig. 14.—Diagram illustrating intention tremor.

A is the point of rest. *A–B* is still the point at rest. *B* is the point where the patient begins to perform an action. *C* is the point where action ceases and the patient

again comes to a point of rest. During repose, line *A–B*, there is absolutely no tremor. When the patient begins to make a movement at *B*, tremor begins, continues, and increases during the continuance of the movement *BC*.

List of diseases in which the intention tremor occurs:

1. Multiple sclerosis.
2. Tumor of crura cerebri.
3. Tumor of the quadrate bodies.
4. Cerebellar tumors.

The Tremor of Paralysis Agitans.—The distribution of the tremor is generally the same; it begins in the fingers and small muscles of the hands, then gradually extends up the arm, skips the trunk, and soon involves the leg and foot. The opposite side is not affected until much later in the disease. The result is that the patient presents a tremor in character hemiplegic. So distinctive is this characteristic that the condition often resembles a real hemiplegia, or a posthemiplegic tremor strongly suggests paralysis agitans. The main point to remember is that the posthemiplegic tremor is decidedly less coarse and faster.

The tremor of paralysis agitans is the slowest of all. The oscillations do not exceed six a second. Their range of movement is very slight. According to Sir William Gowers, they average from one-eighth to three-quarters of an inch. This tremor is very regular and very coarse. The movements consist of flexion and extension, then in a most rhythmic manner come abduction and adduction.

A very marked feature of this tremor is that it is most pronounced, most violent, during repose; any

movement or any action on the part of the patient reduces very greatly the trembling. Thus, it is not at all uncommon to see a patient, who has been trembling violently, raise a glass of water to his lips and drain the contents without spilling a drop.

The handwriting of these patients is, strange to say, not affected till late in the disease. The formation of the letters is slow, but fairly good. The strokes of the pen display fine, wavy irregularities. There is nothing of the jerky appearance characteristic of the intention tremor. The walk of these patients is so characteristic that it has been given a special name. A full description of this gait, known as "festination," is to be found in the chapter on Gaits.

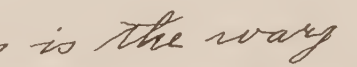


Fig. 15.—Handwriting of a patient suffering from paralysis agitans.

Still another characteristic of this tremor is the fact that it may often be checked for a few seconds by the patient's will, or by his putting the muscles of the hand under great tension. It diminishes in inverse proportion to the patient's attention. It ceases during sleep and is very considerably increased under emotion or excitement. Wilkinson says that it is diminished during a railway journey. As a result of this, one of the forms of treatment has been the ingenious suggestion of a vibrating chair. Weir Mitchell first called attention to the fact that by seizing the index-finger and over-extending it for a short time the tremor can be stopped. Great sorrow or great pleasure will equally augment the tremor. The attitude which the sufferer presents is

unusual; the shoulders are rounded, the back is curved, he leans forward, and the legs are bowed at the knees, giving the appearance of advanced old age. There is marked rigidity, with slowness and deliberation in all movements. The hands, usually the most common seat of the tremor, present a characteristic appearance—one of flexion, immobility, and close approximation of index-finger to thumb. The presence of a regular, coarse tremor occurring in a hand held in a position of flexion corresponding to that of a penman gives rise to a peculiar motion. This motion has been compared to that of crumbling bread, and is designated as "pill-rolling."

This tremor is one of the cardinal signs of the disease known as "paralysis agitans." The others are the attitude, the gait, the mask-like face, and the age. So prominent a position does the tremor occupy in the symptomatology of the disease that the laity have named the condition "the shaking palsy." It is a tremor which has many distinguishing characteristics.

These two tremors in respect to each other stand in opposite extremes. Each is coarse; one is increased on movement, the other decreased. One, the intention tremor, is most common in the young; the other, the tremor of paralysis agitans, is an instance of beginning senility.

The third place of importance can be assigned to the **toxic tremors**. These are tremors caused by poisons, the result of absorption of toxins. They are numerous and varied. The easiest classification is the one which groups them under two headings:

1. Endogenous toxic tremors.
2. Exogenous toxic tremors.

1. The endogenous tremors are those which result from poisons originating inside the body. These are the tremors of *Graves's disease*, *general paralysis*, and *asthenia*.

2. The exogenous tremors are those which result from poisons introduced into the body from without. These are:

(*a*) Alcoholic tremors.

(*b*) Metal tremors.

(*c*) Drug tremors.

The tremor of *Graves's disease* is distributed to the hands and arms. In aggravated cases it may rarely involve the other parts. It never involves the muscles of the face, trunk, or lips. It is fine and constant, numbering about 8 to 10 oscillations per second. Rest has a beneficial and marked effect upon it. Voluntary movement does not necessarily increase it, although any action and movement on the part of the patient tends to increase it. The patient can raise a glass of water with only a slight change of level; it is never spilled. The handwriting in mild cases will show scarcely any abnormality; in the more advanced cases there is evident a slight, wavy irregularity. The walk shows nothing.

It is the tremor which is greatly aggravated by mental exhilaration and excitement. It advances along with the disease. As the latter becomes more pronounced, the tremor becomes more aggravated.

The cardinal signs of Graves's disease are exophthalmos, tachycardia, enlarged goiter, and a fine tremor.

The distribution of the tremor in general paresis is variable: it may involve any muscle; it may involve any group of muscles; it usually involves the muscles of the

face, tongue, lips, and arms. The rate may be fast or slow; therefore, the tremor may be either coarse or fine; the most common type is coarse. The rhythm may be regular or irregular, constant or inconstant. Usually it is irregular and inconstant. The effect of rest is not to stop, only to modify. It should, however, not be forgotten that the tremor of general paresis may assume the

Fig. 16.—Exophthalmic goiter.

form of an intention tremor. Voluntary movement serves to accentuate and aggravate the tremor. The patient raises a glass of water in either a tremulous or jerky manner; if the disease is far advanced he spills the water, but usually only shakes the glass. The ability to draw a straight line is invariably impaired; the paretic handwriting is typical. The sentence begins with large letters

and ends in very minute and indistinguishable hieroglyphics. There is a distinct upward slant to the line, and a tremor is evident on the upstroke of each character. External and emotional causes exert a great influence. In no tremor is this more evident than in that of general paresis. Any excitement, any stimulation, aggravates this tremor. The cardinal signs of general

Fig. 17.—Exophthalmic goiter.

paresis are of two kinds, physical and mental. On the physical side are the unequal, irregular, and immobile pupils; the abnormal reflexes, either absent, exaggerated, or unequal; the tremor, and the ataxia, both of speech and of movement. On the mental side are either the depression or exhilaration, the irritability, the lack of orientation, and the dementia.

Alcoholic Tremor.—The tremor of alcoholism affects the lips, tongue, face, and hands. It may, in aggravated and long-standing cases, involve the rest of the body. It is fine, but slow. In rhythm it is regular and constant. Rest reduces it, while voluntary movement augments it. A marked feature is the fact that it is more in evidence early in the morning than at any other time of the day. Food and drink have a temporary effect of reducing it. In aggravated cases the hands may be so severely affected as to interfere altogether with the patient's ability to raise a glass of water without spilling. The

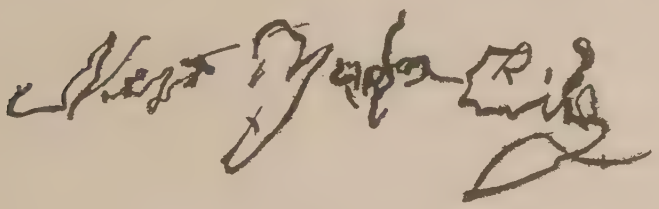


Fig. 18.—Handwriting of an alcoholic.

handwriting is tremulous and uncertain, but the patient never omits words or syllables and never adopts the slanting writing so characteristic of paresis. External influences aggravate it. The walk of the alcoholic is fully described in the chapter on Gaits. The cardinal signs of alcoholism are the tremor, the gait, the dilated pupil, the odor of alcohol, and the facies, bloated, cyanosed, and pasty.

Lead Tremor.—The distribution of the tremor in lead poisoning resembles somewhat that of the tremor in alcoholism; it is more apt to affect the muscles of the hand and face. It may, however, invade the muscles

of the throat and tongue. It may be coarse or fine: usually it is fine, and occurs in the form of fibrillary twitchings. This tremor is exceedingly rare and has no other marked characteristic. The cardinal signs of lead poisoning are the colic, the blue line on the gums, the paralysis, and the presence of lead in the blood.

The mercurial tremor is one of the exceptions to the general rule of toxic tremors. It is coarse and jerky.

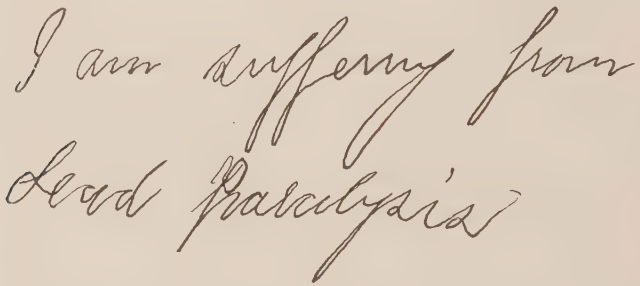


Fig. 19.—Handwriting of a patient suffering from lead tremor.

In the beginning of the toxemia it is present on movement, ceasing during rest. Toward the end, however, it is constantly present. The mercurial tremor is a rare condition, not necessarily because the symptom occurs rarely in the poisoning, but because poisoning by mercury, outside of two or three particular occupations, is met with rarely. It occurs especially among mirror-makers, hat-makers, and those who are employed in working silver mines. The distribution, like that of most toxic tremors, is to the face and tongue, later to the extremities and entire body. It is supposed to

resemble the intention tremor. It, however, does not, being coarser and more inconstant.

Under the topic of *toxic tremors* mention should be made of the fine tremor which occurs in the later stages of morphinism, in the case of the habitual tea and coffee drinker, and lastly in the case of the cigarette smoker.

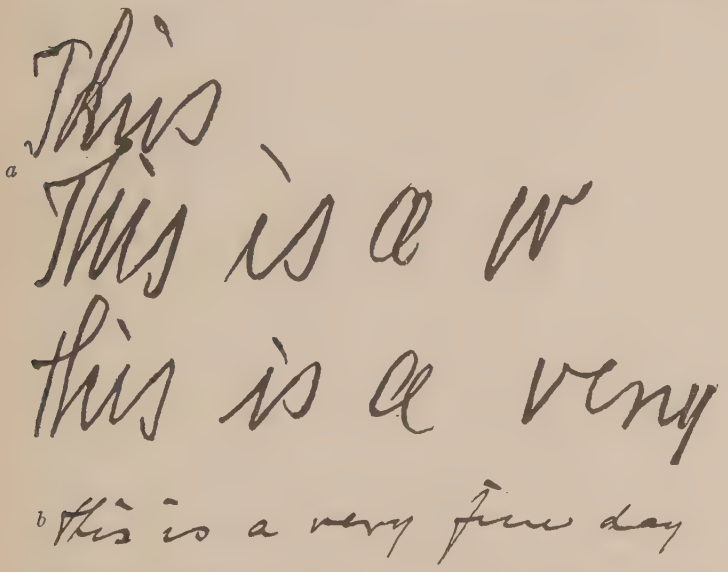


Fig. 20.—*a*, Specimen of handwriting of a patient suffering from chloral and veronal poisoning; *b*, specimen of same handwriting three weeks after stopping all drugs.

These tremors are all distributed to the hands, especially to the fingers and small muscles of the hands. They are best illustrated by having the patient extend the fingers, or, in the more sensitive cases, by having the examiner place his finger-tips extended and touching those of the patient. Chloral and cocain also present tremors.

Asthenic tremor is the name applied to the trembling which accompanies prolonged illness in acute and wasting diseases. Its distribution may involve the entire body, usually, however, only the hands and the wrists. The rate is rapid and fine, the rhythm fairly regular. Rest modifies but does not stop it, while voluntary movement makes it more evident. The patient, as a rule, is too weak either to raise a glass of water, draw a straight line, or walk. If he is strong enough to write, the letters are faulty and very wavy. Any outside influence aggravates it. It is most apt to occur in phthisis, in heart disease, and following typhoid.

Posthemiplegic Tremor.—The posthemiplegic tremor is distributed over one-half the body, is slow, regular, and coarse. It resembles in appearance the tremor found in paralysis agitans. Unlike that of paralysis agitans, it is diminished by rest and aggravated by voluntary movement. In the aggravated cases the patient is unable to hold a glass of water or to write. The gait is, of course, hemiplegic. Excitement or emotion greatly aggravates it.

Tremors from Gross Brain Lesions.—The tremors from gross brain lesions are most apt to involve the arm and leg. They may be fine or coarse, and generally are regular. Rest diminishes but does not stop them, while voluntary movement increases them. The patient raises a glass of water, writes, or walks with great difficulty. The effect of external or emotional influence is slight, except in those cases in which the tremor is hemiplegic in character. The cardinal signs of the accompanying disease are headache, blindness, vertigo, and increasing paralysis. The tremor accompanying tumors of the crus

and pons is coarse and jerky, while that accompanying hemiplegia is usually fine.

Cerebellar Tremor.—Cerebellar tremors generally affect the head and upper extremities. They are most apt to be slow and coarse, and sometimes of the intention type. Neither rest nor voluntary movement seem to have any appreciable effect upon them. They are fairly frequent in occurrence. Wyllie says that a tremor is present in 50 per cent. of the cases. He also calls attention to the fact that the tremors are far more frequent when the lesion is on the left side.

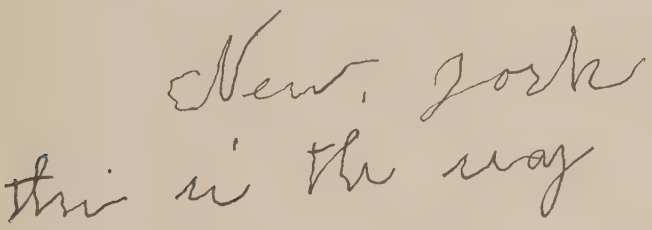


Fig. 21.—Handwriting of a patient suffering from tic.

Hysteric Tremor.—There are hysteric tremors just as there are other hysteric signs. Their distribution may be general, but is most apt to affect the fingers and hands and particular groups rather than particular muscles. They may assume any one of the various types, fine, coarse, intention. Krafft-Ebing has called attention to some in which the tremor resembles that of paralysis agitans. In rhythm they may be regular or irregular. Rest has a marked effect in quieting their motion, and under sleep they are apt to disappear. They are, as one would naturally suppose, much influenced by excitement, fatigue, and emotion. Voluntary movement aggravates

them. The patient raises an object with a distinct tremor and writes in an irregular manner. The principal characteristic of the hysteric tremors is the tendency to simulate or mimic some of the others. For that reason they are apt to present considerable difficulty in diagnosis, as only the absence of all organic signs and toxemia can make their presence even suspected. They are apt to appear early in life and as the result of some shock.

Neurasthenic Tremor.—The tremor in neurasthenia involves the face and especially the eyelids, lips, and surrounding muscles, less often the hands. It is fine and rapid. The effect of voluntary movement is not considerable, as the patient can carry a glass of water with little disturbance, and the handwriting is not diagnostic. There is nothing unusual about the gait. The effect of an external or emotional influence upon this tremor is to increase it. This form is most apt to occur in one who is overworked or tired.

Emotional Tremor.—The emotional tremor is most apt to involve the extremities, especially the legs and knees. It is slow—unusually so—and fine, but regular. Rest brings it to a standstill, while voluntary movement only serves to accentuate it. The patient evinces but slight trembling when he raises a glass or writes, but shows the greatest difficulty when he attempts to walk. This tremor is most often seen in after-dinner speakers, lecturers, and performers. Fear and emotion are its principal causes.

Hereditary Tremor.—The hereditary tremor is most apt to affect the head and arms, especially the latter. It is rapid, fine, and regular. It is not affected by either

rest or voluntary movement. If the distribution involves the arms, the acts of raising a glass of water or of writing display only a slight irregularity. The gait is normal. External and emotional influences have little effect upon this condition. The hereditary tremor has no discoverable cause beyond the fact that it has

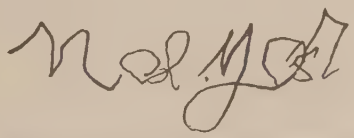


Fig. 22.—Attempt on the part of a patient suffering from Friedreich's ataxia to write New York.

been handed down through several generations. It may appear at an early age—twenty. Graupner has reported the history of a family in which two brothers, a sister, and a niece were all afflicted with a tremor. In these people the distribution was chiefly restricted to the arms. The point to bear in mind in regard to the

Fig. 23.—Specimen of handwriting of Friedreich's ataxia.

so-called hereditary tremor is that it does exist, that it may assume different types in different families, and that, although it is most liable to affect the arms and head, that it may affect any part of the body, and that it may be transmitted through several generations.

Senile Tremor.—In the early stage senile tremor is limited to the head; as it advances, the tremor involves the entire body. It is slow, fine, and regular. Rest does not affect it; voluntary movement always aggravates it, especially as it progresses. The handwriting is irregular and small, and a glass of water is raised in a very slow and tremulous manner. External and emotional influences increase it. The senile tremor develops late in life, rarely making its appearance before the sixth decade. It differs very much from the tremor of paralysis agitans, as in senility the hand moves separately and not with the forearm.

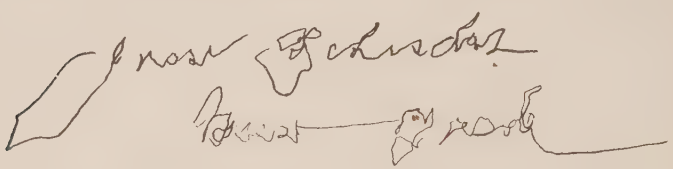


Fig. 24.—Handwriting of a patient suffering from senile tremor.

Simple Tremor.—Apart from all the previous types of tremor there still exist isolated instances in which tremor is the only symptom. It may be fine or coarse, and its distribution is most apt to be in the hands. I have known of an instance where the patient was perfectly well, and was a hard-working professional man in this city. His heredity was excellent. There was absolutely no history of any toxemia or infection. At about fifteen he developed a tremor, coarse and distributed to the right arm. He has now had this tremor for twenty-five years, and at no time in that long period has he had a sick day, or in any way developed any other physical sign or even indication of any nervous or de-

generative condition. There was no hereditary tremor in his family. The characteristic of the simple tremor seems to be that it neither advances nor gets better. Others have reported similar instances, so that there can be no question of the fact that a condition similar to the simple tremor does exist.

The fibrillary twitchings which are seen in some of the wasting diseases are divisions of the fine tremors. These are very fine in character, rapid, and affect certain groups of muscles or certain portions of muscles. These twitchings are symptoms of wasting muscle tissue, and are directly traceable to those muscles whose centers in the anterior horns are degenerating. They therefore occur with great frequency in diseases of the lower motor neuron type. They can best be produced by tapping the muscles.

Fibrillary tremors are fine twitchings which are distributed to a particular portion of the body or to a particular muscle. They are usually devoid of rhythm and are irregular. They are inconstant and not affected by emotional or external influences. External stimulation, in the form of blows and cold, or, better still, slight tapping with the finger, will best produce them. They too indicate a disease in the motor cells which control the muscle.

Fibrillary tremors occur in:

1. Progressive muscular atrophy.
2. Syringomyelia.
3. Chronic poliomyelitis.
4. Tabes.
5. Sciatica.
6. Health following excesses.

7. Emaciated persons under the influence of cold.
8. Neurasthenic persons.
9. Bulbar palsy.

The classification of tremors into two divisions has still further advantages. It enables the student at once to arrange and to arrange systematically all those diseases in which tremor is a symptom. It is possible because the presence of either of these tremors conveys particular and especial information. The following table cites the num-

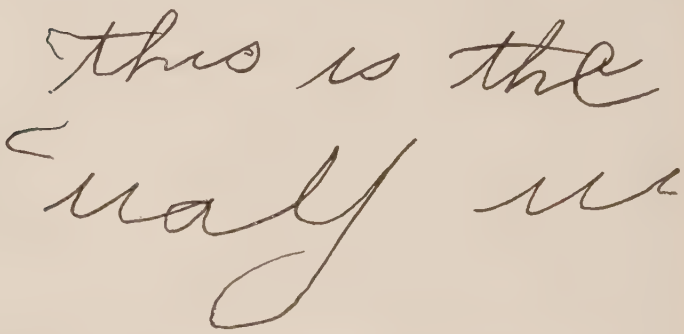


Fig. 25.—Handwriting of a patient suffering from acute chorea.

ber of variations per second of the different types of tremor, as made by different observers:

Paralysis agitans	3 to 7 per second	Wilkinson
Senile	4 to 5½ per second	Dana
Hysteria	5½ to 7½ per second	Mavie
Graves's and alcoholic	8 to 9 per second	Dana
Neurasthenia	10 per second	Dana

A. *A coarse tremor means only one thing*—organic disease.

There are one or two exceptions to this, as the tremor of mercurial poisoning is coarse, and the tremor of

general paresis and hysteria may be coarse or fine. As a general rule, however, the presence of a coarse tremor indicates an organic disease.

The organic diseases in which the coarse tremor appears are: paralysis agitans; general paralysis; multiple sclerosis; tumors of the brain.

B. *A fine tremor, on the other hand, means two things:*

1. Functional disease.
2. Toxic disease.

The functional diseases in which the fine tremor occurs are neurasthenia and hysteria; and the toxic

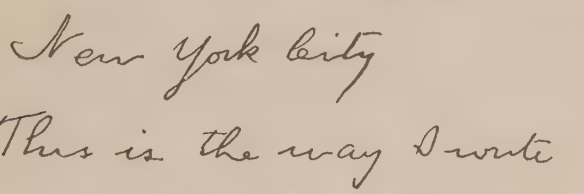


Fig. 26.—Handwriting of a patient suffering from Huntington's chorea.

diseases are Graves's disease, alcoholism, metallic poisoning (except mercury), drug poisoning.

Division of Tremors as Regards Repose and Movement of the Limbs (Wilkinson)

1. Tremors present during repose ceasing during movement (tremor of paralysis agitans).
2. Tremors ceasing during repose present during movement (tremor of multiple sclerosis; brain lesion; others at a very early stage).
3. Tremors present during repose exaggerated during movement) senile; asthenic; hysteric; alcoholic).

CHAPTER V

TROPHIC DISORDERS

The word trophic is from the Greek τροφή, and signifies nourishment or nutrition. The trophic disorders are, therefore, disorders of nutrition. They are not diseases, but symptoms—symptoms which may affect any part of the body—the skin, the mucous membrane, the muscles, the joints, and the bones.

The explanation of these disorders is somewhat obscure. They are closely related to the vasomotor apparatus, and no doubt some of them are connected with and modified by the mechanism of the latter. The most probable, as well as the most general causes, however, lie in an interference with the nutrition of the centers. Starr explains it best when he says: "The central gray matter controls the nutrition of the muscles. Under certain conditions the ordinary sensory impulses are interfered with or perverted, and they, lacking their accustomed guide to repair, and misled by normal impulses, produce effects which are needless under the circumstances, or fail to produce those which ordinarily would be required."

There are centers which preside over the nutrition of the various structures of the body. These are called trophic centers. They, together with the peripheral nerve and its termination, are responsible for the nutrition of the skin, muscle, nerve, joints, and bone. Any injury, therefore, which is inflicted upon the cells in the

anterior horn of the spinal cord and its efferent fiber will

Fig. 27.—Charcot joint, showing retroflexion.

be reflected in the nutrition of these various structures of the body.

The distribution of the trophic disorders is of two

kinds. It may be general or local, that is, the disorder may be of such a type as to involve either the entire body or parts of it, or may be so localized as to follow the distribution of a given nerve or root, or some segment of the brain or spinal cord.

I have arranged these trophic disorders under three headings—atrophy, hypertrophy, and dystrophy, and then I have placed in a list under each of these headings the various structures of the body from without inward, as skin, muscles, joints, bones.

Atrophy

The atrophies are as follows:

1. Of the skin:
   - (*a*) Multiple neuritis.
   - (*b*) Scleroderma.
   - (*c*) Leprosy.

2. Of the muscles:
   - (*a*) Progressive muscular atrophy
   - (*b*) Paralyses
     1. Poliomyelitis.
     2. Hemiplegia.
     3. Facial palsy.
     4. Birth paralysis.
     5. Bulbar paralysis.
   - (*c*) Syringomyelia.
   - (*d*) Tumor of the cord.
   - (*e*) Tabes.
   - (*f*) Multiple neuritis.
   - (*g*) Secondary to inflammatory joint disease.
   - (*h*) Injury to nerves or joints.
   - (*i*) Leprosy.
   - (*j*) Hysteria.
   - (*k*) Multiple sclerosis.
   - (*l*) Myxedema.

3. Joints and bones:
   - (*a*) Paralyses in children
     1. Birth palsy.
     2. Cerebral palsy.
     3. Poliomyelitis.
   - (*b*) Cretinism.
   - (*c*) Progressive muscular dystrophy.

4. Of the nervous system:
   - (*a*) Optic nerve.
   - (*b*) Simple atrophy of nerve-fibers after injury to peripheral nerves or tracts, or of tracts in primary spinal cord diseases.

Muscular Atrophy.—Under the heading muscular atrophy a few variations exist which are of sufficient importance to merit mention, and of sufficient value in differential diagnosis to warrant study.

These are progressive muscular atrophy, poliomyelitis, and tumor of the cord.

In *progressive muscular atrophy* the wasting begins in particular groups of muscles. It may be in the muscles of the shoulder, of the neck, or scapula, or those of the hips or legs, but usually it begins in the small muscles of the radial side of the hands. The characteristic of this type is to jump from one group to another—not to progress from one muscle to the next. Thus, the atrophy may begin in the small muscles of the hand, skip those of the forearm and upper arm, and then affect the neck or shoulder group. The condition continues to progress and eventually involves the entire muscular system; not only does it involve the entire muscular system, but also it involves the entire muscular system to a marked degree—to so great a degree that the muscles become mere strands.

The *atrophy in poliomyelitis* differs in many respects from that of progressive muscular atrophy. Its onset is rapid and sudden; it does not jump from one group to another. The most striking difference, however, is in distribution. This follows the segmental involvement in the cord and therefore results in an atrophy involving groups of muscles. As the spinal cord involvement may be extensive or slight, so may the atrophy affect one or several groups of muscles.

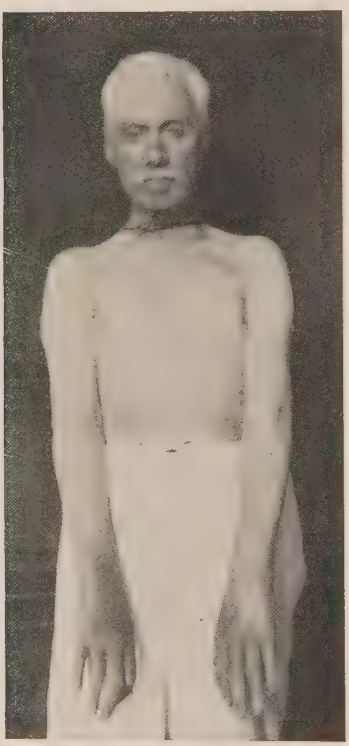


Fig. 28.—Late symmetric atrophies in tabes.

It may affect any group, but most commonly those of the cervical or lumbar enlargement.

The *atrophy in tumor of the spinal cord* is wholly dependent upon the location and extent of the lesion. Inasmuch as all muscles receive nourishment and supply from more than one segment of the cord, the extent of atrophy depends upon the vertical extent of the tumor. The distribution usually involves an entire extremity.

The *atrophies in tabes* are divided by Marie into two

classes: (1) The early and (2) the late. 1. The early forms present small localized and unilateral atrophies, in which there occur fibrillary twitchings and sometimes the reaction of degeneration. 2. The late atrophies are symmetric, appear late in the disease, and do not present the reaction of degeneration.

Bones and Joints.—In both **birth and cerebral palsies** there is an atrophy of the bones and joints. The bones of the paralyzed limb are smaller and shorter, and, as is proved by the *x*-ray, the joints less well developed.

In **poliomyelitis** the same atrophy has been shown. The Haversian system of the bones of the affected side is less well developed, the bones are smaller, and the joints contracted.

The **cretin** displays diminished physical attributes as well as mental. The joints and bones are undersized.

Hypertrophy

1. Of the Skin:
   - (*a*) In many skin diseases, as ichthyosis, etc.
   - (*b*) Elephantiasis.
   - (*c*) Myxedema.
   - (*d*) Syringomyelia.
   - (*e*) Brittle, ridged nails, as in syringomyelia and traumatic neuritis.
2. Of the Muscles and Subcutaneous Tissue:
   - (*a*) Adiposis dolorosa.
   - (*b*) Elephantiasis.
3. Of the Joints and Bones:
   Acromegaly and gigantism.
4. Of the Nervous System:
   - (*a*) Leprosy, in the ulnar nerve.
   - (*b*) Elephantiasis.

Dystrophy or Mixed Atrophies and Hypertrophies and Vascular Lesions

1. In the Skin:

(*a*) Bed-sores:

1. Multiple neuritis.
2. Tumors of the brain.
3. Tumors of cord.
4. Hemorrhage in brain or cord.
5. Syringomyelia.
6. Myelomalacia.
7. Meningitis of brain or cord.
8. Delirium tremens.
9. Any restless state where there is need of restraint.
10. Torticollis (on ear).
11. Fracture of the spinal column.

(*b*) Raynaud's disease:

1. Anemias.
2. Hysteria.
3. Epilepsy.
4. Tabes.
5. Neurasthenia.
6. Myelitis.
7. Insanity, especially acute mania.
8. Urticaria.
9. Angioneurotic edema.
10. Scleroderma.
11. Trauma.
12. Influenza.
13. Grief.
14. Fatigue.
15. Fright.

(*c*) Perforating ulcer of the foot:

1. Tabes.
2. General paresis.
3. Leprosy.
4. Paralysis of sciatic nerve.

(*d*) Gangrene in syringomyelia, neuritis of any form, hysteria.
(*e*) Erythromelalgia.
(*f*) Pemphigus.
(*g*) Edema.
1. Generalized.
2. Localized.
(*a*) Angioneurotic edema.
(*b*) Blue or white edema of hysteria.
(*c*) Localized areas of hard edema.
(*h*) Change in color of the hair:
1. Generalized.
2. Localized, as in change in one eyebrow, or in beard following neuralgia.
(*i*) Zoster.
(*j*) Keratitis:
1. Sciatica.
2. Spondylitis deformans.
3. In neuritis of ophthalmic branch of trifacial in trifacial neuritis.
4. Tumors of brain.
(*k*) Whitlows—small abscesses in syringomyelia.
(*l*) Ulcers in mucous membrane of mouth and nose, in trifacial paralysis.

2. In the Muscles:
(*a*) Spinal form of progressive muscular atrophy (the Aran-Duchenne type).
(*b*) Hereditary form (the Werdnig-Hoffmann type).
(*c*) Dystrophy.
(*d*) Erb's juvenile form of dystrophy.
(*e*) Facio-scapulo-humeral type of Landouzy-Déjérine.
(*f*) Peroneal or leg variety of Charcot-Marie-Tooth.
(*g*) Myotonia congenita—Thomsen's disease.

3. In the Joints and Bones:
(*a*) Arthropathy or Charcot joint in tabes and syringomyelia.

(*b*) Spontaneous fractures and osteoporosis in tabes, syringomyelia, and progressive muscular dystrophy.
(*c*) Scoliosis in syringomyelia.
(*d*) Gigantism in pituitary disease.

Fig. 29.—Charcot knee, showing abduction at knee-joint.

(*e*) Falling out of teeth in tabes, trifacial palsy.
(*f*) Joint swelling in multiple neuritis, myxedema, and hemiplegia.

4. In the Nervous System:

(*a*) Choked disc.
(*b*) Myelitis, encephalitis, and compression myelitis.
(*c*) Neuroretinitis.

Perforating ulcer of the foot is one of the common trophic changes. It is one of the leading symptoms of tabes. Usually it is located in the plantar surface of the foot or the heel. It begins as a small corn. There is a slight discharge; a sinus is formed, and eventually a suppurating tract leads down to the bone of the joint. It is painless. The condition is almost impossible to heal. A perforating ulcer may also affect the mouth or the region of the buttocks.

Diseases in Which Perforating Ulcer is Found

1. Tabes.
2. Syringomyelia.
3. Leprous neuritis.
4. Injuries to posterior tibial nerve.
5. Traumatic neuritis of sciatica.
6. Tumor of the cord (sacral region).
7. Progressive muscular atrophy (Charcot-Marie-Tooth).

Aran-Duchenne is the name applied to that particular group of cases of progressive muscular atrophy in which the small muscles of the hand are first affected. Later the condition spreads and finally involves the entire body. The affected muscles show the characteristic symptoms of fibrillary twitchings, wasting, and reaction of degeneration.

Werdnig-Hoffmann is the name applied to that type which begins in infancy and rapidly proves fatal. It involves first the muscles of the hip, then those of the back and abdomen, and finally those of the neck and shoulders. It is sometimes called the infantile type.

Erb's juvenile type is the name applied to that variety which appears in youth and tends to affect several members of one family. It first involves the arms and shoulders.

Landouzy-Déjérine is the name applied to that variety

Fig. 30.—Progressive muscular atrophy (Aran-Duchenne type).

which affects the face as well as the arm and shoulder muscles. This is the one type in which the facies present a characteristic appearance.

Charcot-Marie-Tooth is the name applied to that variety which affects the peroneal muscles. From the peroneal

group it advances upward, involving first the muscles of the calf, then of the thighs, and finally may extend to the trunk and arms.

Myotonia congenita is the name applied to a rare condition first described by Dr. Thomsen. It is characterized by the development of tonic cramps when the patient attempts any voluntary movement. It is also a hereditary family condition.

Trophic disorders of the joints may be of several types. The larger joints are those most apt to be affected, the knee and shoulder especially. These disorders may extend from a slight affection to the typical *Charcot joint.* They are found more often in female patients than in male. The most common and usual type is first a slight effusion without pain, from which the patient recovers, to be followed after a few weeks by another effusion, somewhat larger and more lasting, which then shows a weakness of the muscles and structures, and then, with the aid of the *x* ray, demonstrates a complete disintegration and destruction of the joint, surface lining, and structures. Charcot was the first to describe these joints. He divided them into two classes—the atrophic and the hypertrophic. In each the destruction of the joint is complete. The fluid is, as a rule, serous, but in a few cases may be mixed with blood. The *x*-ray is of considerable value in diagnosing these arthropathies, and should always be used; it enables the examiner to differentiate between the simple effusion and a typical Charcot joint. In the latter the *x*-ray shows the destruction of all the tissues, including the bone, and admirably displays the condition which Church describes as a "bag of bones." These Charcot joints

Fig. 31.—Charcot wrist in tabes.

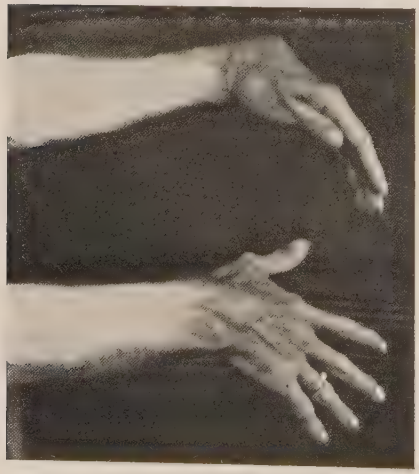


Fig. 32.—Charcot wrist in tabes.

are typical of locomotor ataxia and syringomyelia. In the former disease they assume enormous proportions. Though the larger joints are those most commonly affected, those of the foot, the fingers, wrist, and elbow may be involved. The mobility of the joint approaches the type of pivot joint, and the swing is always painless. The starting-point of these Charcot joints is difficult to indicate, although a careful examination often shows a slight traumatism near the joint involved. Therefore, always bear in mind that a sufferer from a spinal cord disease ought to take special care not to injure, even in the slightest way, an extremity or a joint.

In hemiplegia, in addition to these Charcot joints, slight swellings appear. They differ from the typical Charcot joint in being painful and reddened. They neither appear with such rapidity nor reach such enormous dimensions. Finally, the hemiplegic joint changes are not destructive.

Hysteria presents joint changes. These differ from the Charcot joints and the hemiplegic joints in that they are exceedingly painful. There is rarely any effusion.

The Trophic Changes in Bones.—The trophic changes in bones can be classified under three headings:

1. The tendency to interference with the growth of bones.
2. The hypertrophy of bones.
3. The tendency to fracture.

The hypertrophy is especially illustrated in conditions of acromegaly; the tendency to fracture of the bone is seen in cases of tabes and syringomyelia. Here there is an unusual brittleness, so that a misstep or undue strain may often precede a fracture of one of the long bones.

CHAPTER VI

DIAGNOSIS AND LOCALIZATION OF DISEASES OF THE SPINAL CORD

I. Clinical groups of spinal cord diseases.
II. Symptoms of spinal cord diseases.
III. Anatomical diagnosis.
    1. Spinal localization in the vertical direction.
        (*a*) Motor.
        (*b*) Sensory.
        (*c*) Reflexes.
        (*d*) Trophic.
    2. Spinal localization in the transverse direction.
IV. Pathological diagnosis.
V. Regional diagnosis.
VI. Transverse lesions of the spinal cord.
    1. Incomplete.
    2. Complete.
    3. Posture.
VII. Unilateral lesions.

DIAGNOSIS AND LOCALIZATION

To understand the diagnosis and localization of diseases of the spinal cord it is best first to combine spinal cord diseases into certain definite clinical groups. I have, therefore, adopted the classification of Williamson and made four groups: (1) in which the prominent symptoms are those of a transverse lesion of the cord; (2) of atrophic paralysis; (3) of a spastic paraplegia; (4) of ataxia.

Under these four different headings are grouped practically all diseases of the spinal cord.

Following this grouping I have enumerated a list of the symptoms of spinal cord diseases.

The next three sections of this chapter deal with the anatomical, pathological, and regional diagnosis. By anatomical diagnosis I mean spinal localization; by pathological diagnosis I mean the nature of the lesion, whether inflammatory, degenerative, or resulting from pressure; by regional diagnosis I mean the relationship which the external bony landmarks of the spinal column bear to the spinal segments.

I have then devoted two sections respectively to transverse lesions of the spinal cord and to unilateral lesions of the spinal cord.

CLINICAL GROUPS OF SPINAL CORD DISEASES

1. Diseases in which the prominent symptoms are those of a transverse lesion of the cord:

(1) Spinal hemorrhage.
(2) Apoplectiform myelitis.
(3) Acute transverse myelitis.
(4) Intramedullary tumor.
(5) Extramedullary tumor.
(6) Vertebral tumor.
(7) Vertebral caries.

2. Diseases in which the prominent symptoms are those of atrophic paralysis:

(1) Anterior poliomyelitis.
(2) Muscular atrophy.
(3) Amyotrophic lateral sclerosis.
(4) Syringomyelia.
(5) Cervical pachymeningitis.

(6) Injuries and diseases of nerves.
(7) Muscular dystrophy.

3. Diseases in which the prominent symptoms are those of a spastic paraplegia:

(1) Compression myelitis.
(2) Fracture-dislocation.
(3) Gunshot and other wounds of the cord.
(4) Acute transverse myelitis and disseminated myelitis.
(5) Hematomyelia and meningeal hemorrhage.
(6) Spinal meningitis and pachymeningitis.
(7) Syphilitic myelitis, meningomyelitis.
(8) Amyotrophic lateral sclerosis.
(9) Disseminated sclerosis.
(10) Syringomyelia.
(11) Combined posterolateral degeneration and sclerosis, associated with anemia and various toxic conditions.
(12) Erb's syphilitic spinal paralysis.
(13) Occasionally general paralysis of the insane.
(14) Cerebral diplegia (Little's disease).
(15) Primary lateral sclerosis and hereditary spastic paraplegia.

4. Diseases in which the prominent symptoms are those of ataxia:

(1) Locomotor ataxia.
(2) Friedreich's disease.
(3) Ataxic paraplegia.
(4) Combined posterolateral degeneration associated with anemia and various toxic conditions.
(5) Some forms of spinal syphilis.
(6) Occasionally disseminated sclerosis.

SYMPTOMS OF SPINAL CORD DISEASES

1. Paralysis.
2. Changes in the reflexes.
3. Disturbances in the control of the sphincters.
4. Alterations of gait.
5. Defects of sensation.
6. Ataxia.
7. Pain.
8. Trophic disorders.

ANATOMICAL DIAGNOSIS

The anatomical diagnosis in spinal cord diseases means the localization of the lesion. It is, therefore, divided into two headings:

1. The spinal localization in the vertical direction.
2. The spinal localization in the transverse direction.

1. **Spinal Localization in the Vertical Direction.**—Correct localization in the spinal cord necessitates an accurate knowledge of two important points and two subordinate points. The two important points are the motor functions and the sensory functions, as controlled by the segments of the spinal cord, and the nerve-roots running to the spinal cord. In other words, it is necessary to understand that the cord is divided into segments, which segments are bounded both anteriorly and posteriorly by a pair of roots. The anterior nerve-roots supply motion to certain muscles. The posterior nerve-roots supply sensation to certain portions of the skin. If, therefore, we can ascertain accurately and definitely just what muscles are paralyzed, and, secondly, just what areas of the skin are anæsthetic, we can then trace back to the cord the origin of these supply roots both in the anterior and posterior, and in that way we can definitely

localize the seat of the injury. It must be borne in mind, however, that each muscle derives its nerve-supply not alone from one particular segment, but also from the segment immediately above and from the one immediately below the main source of supply. The same holds true of the innervation or supply to the skin. Therefore, in order to have a complete paralysis of the muscles, and to have a complete loss of sensation in the area of the skin, it would be necessary to have a destruction of not only one segment of the cord, but probably of three, the one in which the supply was most pronounced, the one immediately below, and the one immediately above. In addition to these two important points there are two other subordinate factors which enter into the problem of localization in the spinal cord. These two subordinate points are the state of the reflexes and the condition of muscular atrophy. It is almost impossible to remember the exact supply of each muscle, and therefore in spinal cord localization it will be necessary to have a table which will give on one side the names of the spinal segments and on the other side a list of the muscles supplied. The list on p. 111 is taken from the best authorities and should be memorized.

The relation of the posterior roots to the innervation of the skin is somewhat irregular and not sharply defined. It therefore requires some slight description. In the first place, areas of pain or of anæsthesia occupy different shapes on the trunk from that which they occupy in the extremities. On the trunk the areas of skin supplied by a particular nerve-root run around the body in a circular axis, whereas on the legs and arms these areas run lengthwise, that is to say, parallel with the extremity.

Motor Localization

Nerve Root.	Muscles.
Cervical	4. Diaphragm, scaleni, supraspinatus, infraspinatus (? rhomboids and teres minor).
	5. Biceps, deltoid, brachialis anticus, supinator longus, supinator brevis (? coracobrachialis).
	6. Subscapularis, pronator teres, pronator quadratus, teres major, latissimus dorsi, pectoralis major (in part), triceps, serratus magnus (? pectoralis minor).
	7. Extensors of wrist (+ ? extensors of fingers).
	8. Flexors of fingers (+ ? flexors of wrist).
Dorsal	1. Small muscles (intrinsic muscles) of hand, oculopupillary fibers.
	2.–10. Intercostal muscles.
	11.–12. Abdominal muscles.
Lumbar	1. Quadratus lumborum.
	2. Cremaster.
	3. Sartorius, adductors of thigh, ileopsoas.
	4. Extensor quadriceps cruris, abductors of thighs.
	5. Flexors of knee (hamstring muscles).
Sacral	1. Calf muscles.
	2. Glutei, peronei, anterior tibial muscles, intrinsic muscles of foot.
	3. Muscles concerned in erection of the penis and ejaculation. Centers for erection and ejaculation.
Conus Terminalis	
Muscles of sexual organs, bladder, rectum.	S. 4. Levator ani and centers for sphincters of anus and bladder.

[Taken from Williamson's "Diseases of the Spinal Cord."]

The areas are not sharply defined and, as a rule, overlap each other. This is due to the fact that the skin supply is derived from more than one spinal segment. The diagram (Fig. 33) illustrates this and gives our present knowledge of the sensory innervation. In mapping out the sensory areas of the skin it must not be forgotten that while they run in a circular direction around the body, they do not run quite parallel to the

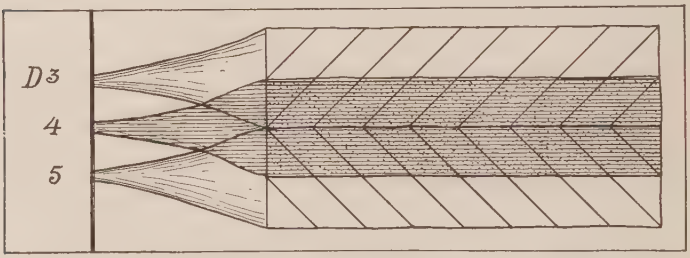


Fig. 33.—Diagram showing overlapping of sensory areas of skin (modified after Sherrington). To the left are three sensory nerve-roots entering the spinal cord. The skin area supplied by root 4 is shaded. The areas supplied by roots 3 and 5 are marked by oblique lines. Note that the upper half of the sensory distribution of root 4 is overlapped by the area of distribution of 3; its lower half by the area of distribution of 5. (Taken from Williamson's "Diseases of the Spinal Cord.")

ribs, but in more of a horizontal direction, beginning a little higher in the back and sloping down, as they reach the front.

The relation of the reflex functions in connection with spinal cord localization necessitates a knowledge of the following facts:

1. The abdominal reflex is supplied by the eighth to the twelfth dorsal roots.
2. The cremasteric reflex, by the first and second lumbar roots.

3. The knee-jerks, by the second, third, and fourth lumbar roots.
4. The Achilles tendon jerk, by the fifth lumbar and first sacral root.
5. The plantar reflex, by the first and second sacral roots.

Increased susceptibility to sensation is a frequent development in those cases in which there is only partial involvement of the root, therefore its presence can be taken as a guide to the upper limit of the lesion. This again is due to the fact that the innervation of any particular part of the skin is derived from more than one segment. The part in which the sensation is increased, therefore, derives its supply from the upper limit of the lesion. In studying this question of spinal cord localization it is of the greatest importance to know—

1. The relation of the spinal cord segments to the vertebræ.
2. The area of origin of the roots.

To examine for spinal cord localization it is necessary to have a knowledge of—

1. The symptoms of spinal cord disease; to examine for each, and to corroborate or eliminate its presence.
2. The level of the involvement of the anterior motor root zone.
3. The level of the involvement of the sensory or posterior root zone.
4. The attitude and posture of the patient.

In trophic disturbances ascertain what particular muscles are atrophied. Particular muscles receive their supply from particular segments of the spinal cord, there-

fore, if the atrophic muscles can be singled out, it will be possible to locate the exact portion of the spinal cord in which the lesion is situated.

Usually, however, the atrophy is not restricted to a single muscle, but involves a group. This arises from the fact that the cells supplying a group of muscles are located close together in one particular spinal segment.

2. **Spinal Localization in the Transverse Direction.**—The spinal cord is made up of tracts, columns, and horns. These run up and down, transmitting different and various impulses. In disease or injury one or several of these tracts, columns, or horns may be affected. Each contains a different set of fibers, each transmits a different impulse, therefore, according to the tract, column, or horn involved different clinical symptoms develop. The student must know not only the names of these tracts, columns, and horns, but also their functions.

Lesions of the pyramidal tracts produce motor paralysis, but of the upper motor neurone type, of the corticospinal type. The clinical signs in the paralyzed area are those of spastic paraplegia, that is, rigidity, exaggerated reflexes, paralysis, contractures, and increased muscle tonus. The pyramidal tracts degenerate downward and, therefore, the motor paralysis is below the level of the lesion.

Lesions of the posterior columns produce sensory disturbances, especially as to pressure and muscle joint sense. All types of cutaneous impressions are delayed and there is a diminution of the sense of temperature and of pain. The clinical signs are ataxia, loss of deep reflexes, and paresthesiæ. These posterior columns de-

generate upward and, therefore, the signs occur above the level of the lesion.

Lesions of the anterior horns produce motor paralysis, but of the lower motor neurone type, of the spinomuscular type. This paralysis is restricted entirely to the particular group of muscles whose cells of supply in the anterior horn are affected, and usually in lesions of the anterior horns instead of there being a complete extremity involved, or one isolated muscle, there will be a group of muscles. The clinical signs are:

1. Atrophy.
2. Loss of reflexes.
3. Vasomotor paralysis.
4. Fibrillary twitchings.
5. Reaction of degeneration following electrical examinations.

Lesions of the posterior horns result in sensory disturbances or anesthesia in the area corresponding to the spinal cord supply.

Lesions of the posterior roots, if complete, cause anesthesia; if not complete, severe, irritating pain and hyperalgesia.

Lesions of the anterior roots produce the same results as lesions of the anterior horns.

Lesions which occur either in or immediately around the central canal produce a set of symptoms peculiar to themselves. Sensations of pain and heat are destroyed, while those of touch and contact are unaffected. In addition to this, disturbances of the joints occur. The best illustration of lesions in this region occur in syringomyelia.

Lesions which involve only one lateral half of the cord cause a so-called Brown-Séquard set of symptoms.

It is, of course, possible to conceive of a condition in which two or more of these tracts are similarly and symmetrically affected. The best illustration of this occurs in amyotrophic lateral sclerosis, in which the lesion is both in the lateral columns and the anterior horns, the resultant clinical symptoms being rigidity, contractures, and a progressive muscular atrophy. Another illustration is that which occurs in ataxic paraplegia. Here the lesion involves both the motor tract and the posterior columns, so that the clinical signs are ataxia, rigidity, weakness, and loss of reflexes.

Some of these cord diseases are symmetrical and involve both sides of the cord, others are unilateral. In the former instance the clinical signs affect equally both legs, both arms, and the two sides of the body; in the latter the clinical signs differ on the two sides.

PATHOLOGICAL DIAGNOSIS

Pathological diagnosis in spinal cord diseases means the nature of the lesion. The three most important types of lesions are:

1. The vascular lesions.
2. Inflammatory changes.
3. Degenerative changes.

Vertebral caries, tumors of the cord, and some forms of spinal syphilis cause a compression myelitis and, therefore, may be placed under the heading of degenerative changes.

The following table copied from Williamson takes up the whole question of pathological diagnosis in a very simple way:

1. Vascular lesions.
   Abrupt—minutes.
      (1) Hemorrhage (spinal and meningeal).
      (2) Puncture wounds.
      (3) Compression from fracture-dislocation,
      (4) Softening from thrombosis (apoplectiform myelitis).
   Acute—hours or days.
      (5) Acute myelitis.
      (6) Acute anterior poliomyelitis.
      (7) Acute syphilitic myelitis.
      (8) Acute meningitis.
      (9) Landry's paralysis.
2. Inflammation.
   Subacute.
      (1) Subacute myelitis.
      (2) Subacute anterior poliomyelitis.
   Chronic.
      (3) Pachymeningitis.
      (4) Chronic meningitis.
      (5) Some forms of spinal syphilis.
3. Degeneration.
   Very chronic—half years or years.
      (1) Amyotrophic lateral sclerosis.
      (2) Progressive muscular atrophy.
      (3) Chronic anterior poliomyelitis.
      (4) Disseminated sclerosis.
      (5) Tabes dorsalis.
      (6) Friedreich's disease.
      (7) Ataxic paraplegia.
      (8) Combined posterolateral degeneration of various forms.
      (9) Syringomeylia.

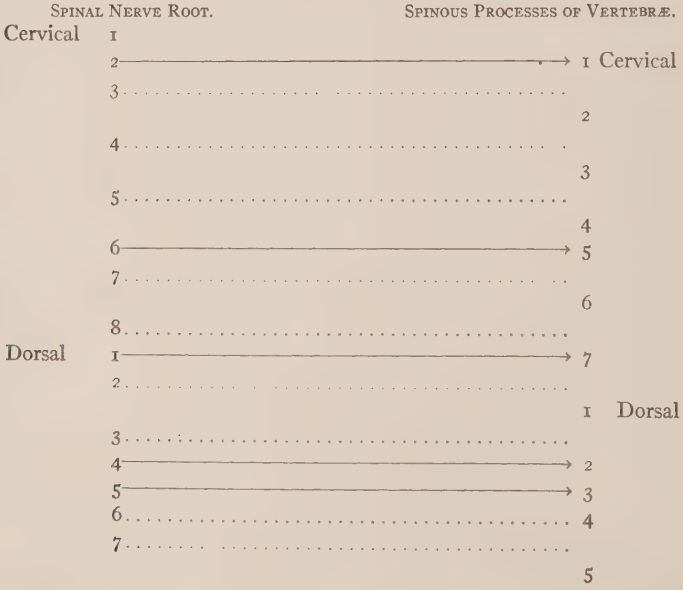
REGIONAL DIAGNOSIS

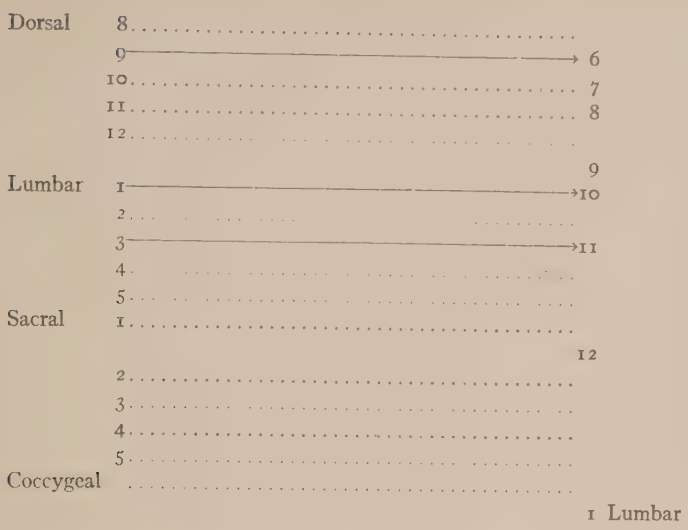
Regional diagnosis is the term which I use to designate the relationship existing between the spinal roots and segments and the spines of the vertebræ. I have compiled a table which gives on one side the superficial origin of the spinal nerve roots, and on the other the spinous process of the corresponding vertebræ. The three important landmarks to remember are:

1. The commencement of the spines of the scapulæ are on a level with the third dorsal spine.

2. The lower angle of the scapula is on a level with the seventh dorsal spine.

3. A transverse line connecting the crest of the two iliac bones crosses the spinous process of the fourth lumbar vertebra.





TRANSVERSE LESIONS OF THE CORD

Transverse lesions of the spinal cord may be of two types:

1. Incomplete.
2. Complete.

1. An *incomplete transverse lesion* of the spinal cord indicates that the whole of the nerve-fibers at the seat of the disease are not destroyed, the lesion is not completely transverse. In this type the symptoms usually are as follows:

1. Paralysis of both legs.
2. Paralysis of bladder and rectum.
3. Impaired or lost sensation in the trunk and legs, the upper limit varying with the level of the lesion. As the condition progresses spastic paraplegia develops.

A lesion in the upper dorsal region adds to the above symptoms a paralysis of the intercostal muscles.

A lesion in the lower cervical region causes a paralysis and atrophy of certain muscles of the arms, a loss of sensation which extends down the inner sides of the arms, according to the level of the lesion. There is, of course, added to these symptoms those which occur in the case of paralysis of the lower dorsal region.

A lesion in the upper cervical region usually adds to the above symptoms a paralysis of the diaphragm, which results in paralysis and death.

A lesion in the lumbar region causes flaccid paralysis of the legs together with an atrophy, and reaction of degeneration, a paralysis of the bladder and rectum, bed-sores, and lost knee-jerks.

2. *Complete transverse lesions* of the spinal cord result in a complete loss of knee-jerks, atrophy of the muscles, loss of electric excitability, loss of sphincter control, and loss of sensation. The characteristics of the bladder in complete transverse lesions are a contraction with constant dribbling.

The one important factor which will help distinguish between complete and incomplete transverse lesions of the spinal cord is the condition of the deep reflexes. In partial transverse lesions the reflexes are *active;* in complete transverse lesions, where the cord is completely divided, the deep reflexes are *lost.*

3. *Posture.*—The following is taken from Starr:

"When the patient is confined to his bed after an injury of the spinal cord, such as occurs from fractures and dislocations, the position assumed in bed differs according to the level of the injury.

"If the sacral segments are crushed, the patient is unable to move the feet and ankles, and hence the feet lie in an extended position, assuming the position of drop-foot. The thighs and knees can be moved in this condition.

"If the lesion involves the lower half of the lumbar enlargement, the patient lies with the thighs drawn up, the legs flexed, and he is unable to straighten the legs voluntarily or to lift the feet.

"If the lesion involves the entire lumbar enlargement, the patient lies with the thighs, legs, and feet extended, and cannot move them.

"If the lesion is in the dorsal region, the lower extremities are more or less completely paralyzed, but there is no tendency to drop-foot, and the limbs offer some resistance to passive motion, the tendon reflexes being exaggerated and the muscles somewhat rigid. If there has been a total destruction of the cord, the muscles are rerelaxed and the tendon reflexes lost.

"If the lesion involves the first dorsal and last cervical segments the hands will be in a position of main en griffe, but the elbows and shoulders can be freely moved.

"If the seventh cervical segment is destroyed, the forearms are partially flexed and lie upon the body with the hands pronated. Voluntary movements of the wrist are impossible, but the elbow and shoulder can be moved.

"If the lesion lies at the sixth cervical segment, the arms are abducted from the side, the forearms are supinated, wrists and fingers being paralyzed.

"If the lesion is at the fifth segment, the arms lie extended and relaxed at the side of the body, all motion being impossible.

"These forced positions are assumed because of the fact that at any level of the cord the muscles controlled at and below the lesion will be paralyzed, and those controlled by the cells just above the lesion will be actively contracted by the state of irritation into which their motor centers are thrown, or if two centers of opposing action are irritated together, the stronger will overcome the weaker, and the resulting position, once assumed, cannot be corrected voluntarily; hence the fixed position, once assumed, is maintained."

UNILATERAL LESIONS

Unilateral lesions of the spinal cord are also known as the Brown-Séquard symptom. This lesion produces one set of symptoms on the side of the lesion, and another set of symptoms on the side of the body opposite to the lesion. The symptoms occurring on the side of the lesion are:

1. Motor paralysis of the leg, with increase of the tendon reflexes, though at first diminished. In time the leg becomes spastic, and then ankle-clonus and the extensor type of plantar reflex develop.
2. Loss of muscular sense (sense of position).
3. Slight elevation of skin temperature.
4. Hyperesthesia of the skin.
5. Near the level of the spinal lesion is a narrow transverse band of anesthesia in the area of distribution of the sensory nerve-root, or roots arising from the diseased portion of the cord. In some cases there is a girdle sensation around one-half of the trunk.
6. Above this anesthetic band is a zone of hyperesthesia.

The symptoms occurring on the opposite side of the lesion are:

1. Anesthesia to pain and temperature, and in some cases to tactile impressions.

2. Motor power, muscular sense, reflexes, and skin temperature normal.

3. Above the anesthesia is a narrow zone of hyperesthesia.

CHAPTER VII

GAITS

In neurology no subject is more interesting and none more brilliant in results than the study of gaits. The text-books ordinarily assign a very small space to the subject; most of them content themselves with saying that the gait is a leading symptom in diseases of the spinal cord. They then dismiss the subject with two or three paragraphs, or, at most, half a page. As a matter of fact, not only is the gait a leading symptom of diseases of the spinal cord, but it also occurs with affections of the brain, peripheral nerves, and even functional conditions. The student will find that he is amply repaid for the time he devotes to this subject.

A man's gait is his manner of walking, together with his carriage. In studying the gait of a patient, therefore, it is necessary to analyze two facts—his walk and his bearing. In analyzing a gait it is necessary first to decide whether the walk and carriage are normal or abnormal; second, if abnormal, in what respects; third, to what distribution the abnormality extends. For instance, a man may be lame in that he drags one foot and holds stiff one arm, whereas another man may be steady in both legs and lurch with his entire body. A gait, therefore, may be abnormal in respect to:

1. The body.
2. The arms.
3. The legs.
4. The feet.
5. The steps.

And in each instance so far as affects the—

(*a*) Attitude.
(*b*) Position.
(*c*) Control.

Before taking up the abnormal gaits, it is first necessary to understand what should be the gait of a normal individual. The normal gait attracts no attention: the body is erect, the head straight, the arms hang gracefully at the side, while the legs move regularly and evenly. The feet are slightly everted; the steps of even, moderate length. The shoes should wear out at the heel, showing that the heel tends to touch the ground first.

The method to pursue in examining a patient's gait is: first, observe his entrance into the room—a patient will walk more naturally on his first introduction than a moment later, when he is conscious of being examined; second, have him walk forward and then backward; third, have him walk in these directions with his eyes shut; fourth, test his ability to stand erect with eyes closed—this is the test to which has been assigned the name Romberg; fifth, have him walk a straight line on the floor, or, in the event of its being a carpet, have him follow the pattern; in this effort one foot should be placed directly in front of the other, and so close that the heel and toe touch each other. This last test is, of

all, the most important. A patient may be able to walk well, so well that even a very careful observer will fail to detect any abnormality. When, however, he essays the final and more delicate test of placing one foot directly before the other so that heel and toe touch, the failure may be absolute. To execute this task successfully a man must be in every sense master of himself. Only the normally gaited man can correctly heel and toe a line. In observing the patient, carry out this test with great care; note carefully the particular crack or line on which the patient begins, as frequently he ends on one considerably to one side of this. In an ataxic gait often the first evidence is that, in attempting to heel and toe a line, the patient will succeed in carrying out the exercise, but will bring up at the end of the room on a line which is wholly different, and considerably removed from the one on which he started.

Another practical way to test a man's gait is to observe the wear of his shoes. One gait will wear down the toe, another the outer side, while the third, from the fact that it has so completely incapacitated the patient, will leave the shoes scarcely worn at all. Observe at which point occurs the greatest friction, and at which point the friction is least.

There are several other tests which may be employed in examining the patient's gait. They are necessary only in doubtful and complicated conditions. The student need not use them in the routine examination, but he should be able to make use of them in emergencies.

Have the patient stand on first his toes and then his

heels; if he is able to do this, have him attempt walking on first the toes and then the heels. The advantage of this test is that it calls into play a particular set of

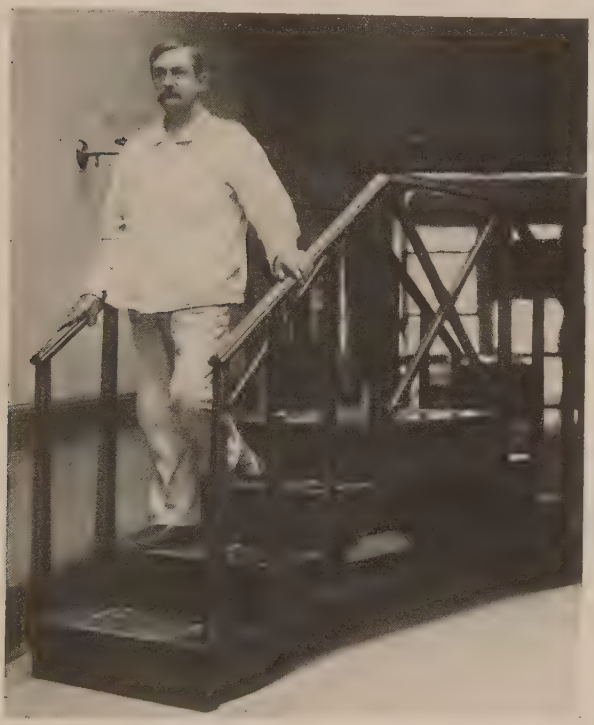


Fig. 34.—Patient practising "stair test."

muscles, first the posterior group of the leg and then the anterior.

Have the patient walk in a circle in both directions and, if possible, test his walking as he goes up and down

a short pair of stairs. This is sometimes called the "stair test" (Fig. 34).

There are three tests which are of considerable aid in distinguishing an ataxic gait and differentiating some of the types of ataxia. These are called Fournier's tests. They are three in number: The first consists of having the patient rise abruptly from the sitting position and at once try to walk. The second is to have him stop abruptly while in the act of walking. The third is to have him turn abruptly while in the act of walking.

The test mentioned above in which one foot is placed directly in front of the other as the patient tries to walk a straight line is sometimes called Fränkel's test.

There are eleven gaits which are important. A list of them follows:

1. The ataxic.
2. The hemiplegic.
3. The steppage.
4. The spastic.
5. The clumsy.
6. The cerebellar.
7. Festination.
8. Staggering.
9. The gait of hysteric conditions.
10. The gait of arteriosclerosis.
11. The normal gait.

1. **The ataxic gait** is very common. It is characterized by an attitude of uncertainty, with a lack of control and a characteristic position of the body. The legs are uncertain. They are under poor control and are held

far apart. This latter posture is followed in order to restore the disturbed equilibrium. The method of using the feet is unusual. They come down with a distinct stamp. The steps are uncertain, and of irregular length. The body sways and seems powerless to control the legs. The position of the head and eyes is unusual, as the patient follows the steps with great care. The legs, therefore, give the appearance of running away with the body. In the advanced cases the patient walks with a cane. In this gait the shoes, as a rule, show little wear. It is well to bear in mind that the lame man whose shoes appear in the best of condition and well kept is usually an ataxic. The attitude of the body is one in which the patient leans forward, usually resting on a stick, while at the same time the entire body sways. This uncertainty of carriage may extend to the upper parts of the body, or may involve only the legs.

The principal features then of the ataxic gait are uncertainty, irregularity, and the stamping step. An ataxic can often be heard before he is seen. He is recognized by his stamp and his stick. The diseases in which the ataxic gait occur are:

1. Locomotor ataxia.
2. Combined posterolateral lesions.
3. Some forms of spinal syphilis.
4. The ataxic type of multiple neuritis.
5. Internal ear diseases.
6. Tumors of the frontal region of the brain.
7. Ataxic paraplegia.
8. General paresis.
9. Friedreich's ataxia.

Fig. 35.—A tabetic reëducating his gait.

2. **The hemiplegic gait** is one of those in which but one leg is affected. The others are the steppage gait and the gait of hysteric hemiplegia. The hemiplegic gait is characterized by the very word to which it owes its name. Hemiplegia means the paralysis of one-half, and the hemiplegic gait is one in which is evident the symptoms, the physical signs, and the characteristics of a paralysis of one-half of the body. The affected leg is rigid. It moves wholly with the body, and not as an independent member. It rotates outward, swinging in a semicircle first from and then toward the trunk. The shoes are worn on the outer side. The affected half of the body is rigid, weak, and paralyzed, so that the patient leans toward the affected side. The arm is held in a characteristic position of semiflexion and rigidity. There is evident a slight asymmetry of the lower two-thirds of the face, with an expression of anxiety and distress. This gait is best described by the word semicircular, or swinging.

It is accompanied by exaggerated reflexes and steps which are regular and uncertain. The toe and whole side of the foot scrape along the floor. The patient often limps. At a distance he presents a twofold appearance of one who is lame and one who is walking in an uneven way, or, rather, in a one-sided way. The excursion of the affected foot is best illustrated by an inverted letter (Ↄ). As in the ataxic gait, the sufferer from the hemiplegic walk can be heard before he is seen. The noise is regular, rhythmic, and grating. The principal features of the hemiplegic gait are rigidity and the side-wheel motion. Other names for the hemiplegic gait are the paralytic

and apoplectic. The conditions in which the hemiplegic gait occur are:

1. Apoplexy, embolus, thrombosis, or hemorrhage.
2. Tumor in the motor area.
3. General paralysis.
4. Paralysis agitans.

3. **The Steppage Gait.**—The steppage gait has many names. It is called the equine gait, the drop-foot gait, and, incorrectly, the alcoholic gait. It is sometimes called the high-step or horse-gait. The cause of the steppage gait is paralysis of the anterior tibial group of muscles; the feet are therefore dropped, the toes drag, and, in order successfully to clear the ground, the patient is of necessity obliged to raise the legs, and especially the knees, sufficiently high to permit the overhanging or drop toes to clear the floor.

The body is not affected. The steps are regular and of normal length; the toes, even when the legs are raised, usually scrape along the floor, or, if

Fig. 36.—Diagram showing excursion of affected foot in hemiplegia. Case of hemiplegia of long duration. The right foot is turned out and drags distinctly (unfortunately the illustration has been retouched and does not show this). The steps are uneven and there is occasional staggering. (From Posey and Spiller's "The Eye and Nervous System," J. B. Lippincott Co., Publishers.)

the patient is unable to raise them sufficiently high, they come down with a rhythmic, flop-like noise. The two legs are, as a rule, equally affected. The wear of the shoes in this gait is unusually characteristic: as a result of the constant scraping and friction upon the toe-pieces, the two shoes are worn out equally at the toe, in a sharp, clean-cut manner, as though cut with a razor. The principal features, then, of the steppage gait are high knee action,

Fig. 37.—Shoes of the steppage gait (worn at toes).

sound of flopping, and characteristic wear of the shoes. The best description of the steppage gait is embodied in the description of the motion of a high-stepping horse. The diseases in which the steppage gait occur are:

1. Multiple neuritis.
2. Nerve lesions involving the nerves supplying the anterior tibial group of muscles.

3. Poliomyelitis.
4. Progressive muscular atrophy.

4. **The Spastic Gait.**—The spastic gait is very difficult for the student to recognize. He invariably confuses it with the ataxic. It is, however, quite different.

The spastic gait rarely affects the legs alone. The attitude of the patient is equally involved. It is a general attitude of stiffness, with a tendency to lean forward. The legs are slightly bent at the knees and decidedly bowed forward. The legs are adducted, with the knees almost overlapping, and the feet inverted; the steps are regular, but very short. The feet can with great difficulty be raised from the floor; the patient hardly walks at all: he shuffles. The feet scrape along the floor, so that the shoes wear out on the front part of the soles, but not at the toe nor in so clean-cut a way as in the steppage gait. The patient is so stiff and incapacitated that he moves slowly, advances with difficulty, and presents the general picture of an old man creeping. This is the gait to which the layman has given the name "creeping palsy."

The distinguishing features of the spastic gait are easy to remember. Each begins, like the word spastic, with the letter "s": stiffness, slowness, scraping, and shuffling. The diseases in which the spastic gait occur are:

1. Primary lateral sclerosis.
2. Disseminated sclerosis.
3. Combined sclerosis.
4. Amyotrophic lateral sclerosis.
5. Hereditary spastic paraplegia.
6. Spastic paraplegia.

7. Syringomyelia.
8. General paresis.
9. Compression myelitis.
10. Spinal meningitis.

5. **The Clumsy Gait.**—The clumsy gait is also known as the sailor gait. Oppenheim admirably describes it by the one word "waddle." The clumsy gait is remarkable for the splendid appearance of the legs. To all outward appearance they possess enormous strength, and display unusual muscular development. Their management develops a very great degree of clumsiness. The attitude of the body is straight; in fact, unusually so. The patient tends to lean backward in order to retain his balance, and the back is hollowed out, with the abdomen unusually prominent; the general carriage is haughty. The muscles controlling the legs are weak and inefficient. As a result of this, those of the trunk are brought into play, and it is they which raise and propel the patient. The gait is, therefore, a rolling one; it reminds one strongly of the efforts made by a man to raise himself by his own boot-straps. The patient rolls from side to side in sailor-like fashion. The steps are a little uncertain, but regular; although in the clumsy gait it could almost be said, with much truth, that there are no steps at all, as the patient progresses entirely from the hips, and with the aid of the trunk muscles.

The principal features of the clumsy gait are the splendid appearance of the legs, together with the clumsy use made of them, and the added aid produced by the muscles of the trunk. The word to remember in connection with this gait is "waddling." The conditions in which the clumsy gait occurs are the muscular dystrophies.

In myxedema sometimes there occurs a slow, clumsy gait.

6. **The Cerebellar Gait.**—To the cerebellar gait is commonly applied the word ataxic. It is a form of ataxia, however, which has distinct characteristics, and a form of ataxia which differs wholly from that seen in tabes and neuritis. The student, therefore, should consider this gait as one apart from, and in no way connected with, the ataxic gait. The cerebellar gait is characterized by great unsteadiness, great irregularity, and a marked tendency to reel to one side. At rest or lying down, the patient does not suffer from ataxia. The moment, however, that he raises himself, he becomes dizzy, and again, when he undertakes to make use of his legs, the infirmity is apparent. The stamping so characteristic of the gait in tabes is absent. The steps are less uncertain, but, if anything, more irregular. On the other hand, in walking, the sway of the body is greater. The best way to emphasize the difference between these two forms of ataxia is by the following statement made by Wyllie: "In the tabetic type of ataxia the legs give the appearance of running away with the body; in the cerebellar type of ataxia the legs give the appearance of being run away with by the body."

Finally, it should never be forgotten that in cerebellar ataxia the patient suddenly, unexpectedly, without any warning, will display the tendency to reel to one side. This is never present in the tabetic ataxia.

This gait is sometimes spoken of as the cerebellar reel or stagger. The patient does reel, and the mode of walking does suggest the stagger seen in the alcoholic more so than in any other gait. It is, however, only confusing to

the student to use the words reel and stagger in describing the cerebellar gait. It is best to keep them for use in describing the gait of the drunkard.

In the cerebellar gait the patient keeps his feet far apart, is very liable to stumble and to run amuck. It is not so much that the legs are uncertain, stiff or irregular, as that the body suddenly and persistently turns to one side, and tends to run away with the legs.

The distribution of the ataxia is, neither in the cerebellar nor in the tabetic type, limited to the legs. In the tabetic type it is, however, apt to involve only the legs; in the cerebellar type, it is more apt to extend to other parts.

The attitude in the cerebellar ataxia is sufficiently marked to warrant studying, and to deserve a name of its own. It has been given the name of the "cerebellar attitude." The head is held high and retracted; there is marked rigidity of the cervical muscles, and one shoulder is higher than the other. If the patient is reclining, the attitude is even more characteristic. He is apt to lie on one side in an attitude of general flexion and rotation, with the head turned laterally, the chin elevated and pointed to one shoulder. The back of the head is, therefore, somewhat lower than the chin. Wyllie, in speaking of the cerebellar attitude, says that in most cases in the standing position there occurs a distinct lordosis; in the reclining position, a distinct tendency to work toward the edge of the bed.

The principal features, then, in the cerebellar gait are the great irregularity, the lateral reeling, and the typical attitude. The diseases in which the cerebellar ataxic type of gait occurs are:

Fig. 38.—Attitude in paralysis agitans showing the leaning backward.

1. Diseases of the cerebellum.
2. Diphtheria.
3. Disseminated sclerosis.
4. Friedreich's ataxia.
5. Diseases of the frontal and quadrate lobes.
6. Diseases of the cerebral cortex.
7. Bulbar softening.
8. Diseases of the crus cerebelli.
9. Myelitis, early stages.

7. **The Gait of Festination.**—Festination is a good English word, but one that is obsolete. It means hastening, hurry. It is derived from the Latin *festinatio*, which means a hastening, haste, or speed. In the description of gaits, it is applied to that particular gait in which the patient makes haste, the gait in which he hurries. In a word, it describes that gait in which the patient practises festination.

In the gait known as festination the patient has one of several characteristic attitudes, in which he leans either forward, backward, or sideways. The legs move more and more rapidly, while the steps become shorter and faster. The general appearance is that of a prematurely old man, bent with age, who is trying, so to speak, to run after his own center of gravity, and is hurrying either forward or backward. Rigidity is the conspicuous characteristic. To so great a degree is the rigidity evident that the patient appears immobile, like one carved from stone and devoid of expression. The folds of the face are obliterated, the play of the muscles absent, the facies statuesque. The man with festination is said to have a mask-like countenance. The shoulders are stooped; the hand presents the appearance of one bent to write, while

Fig. 39.—Attitude of paralysis agitans.

the whole body trembles violently in the advanced cases. The legs, stiff and bent at the knees, are also the seat of a constant and marked coarse tremor. The feet barely leave the ground, so that the steps are of necessity short and shuffling. The patient rises slowly and with great difficulty; he turns deliberately, and for a moment pauses, then he lurches forward, takes his short step slowly, and forthwith begins to hurry. He shuffles, at first slowly, then more rapidly, and at last he runs. This is the gait known as festination. The distinguishing features are rigidity, shuffling, and hurry.

The disease in which festination occurs is paralysis agitans.

8. **The Staggering Gait.**—The staggering gait is preeminently the gait of the alcoholic. It is the gait which one sees in the drunkard on the street. The patient totters, he reels, he leans now forward, now backward, now to one side, and now to the other. He lurches indiscriminately. To the onlooker, at each movement he seems to be on the verge of losing his balance and falling, yet he just recovers himself and rarely, except in the most aggravated cases, does fall. There is almost no control over either body or legs. The feet are unsteady, the steps irregular and uncertain.

The staggering gait invariably carries with it a physiognomy which portrays the drunkard and libertine. This type of gait differs from the cerebellar form of ataxia in that the patient may reel in any one of many directions. It differs from the spinal type of ataxia in that the patient neither follows his feet with the eyes nor presents the stamp which is so characteristic of the gait of locomotor ataxia; it differs from the gait of festination in not having

the rigidity, the tremor, and the characteristic hurry; it differs from the spastic gait in that it lacks the stiffness, the scrape, and the shuffling movements; it differs from the clumsy gait in lacking the heaving or lifting motion, the sailor-like roll, and the marked use of the trunk muscles; it differs from the steppage gait in the absence of any paralysis, any high steppage, and any drop-foot. There is no characteristic wear to the shoes of the drunkard. The principal features, then, of the staggering gait are the wide diversity of excursions, the marked reeling, and the great degree of general ataxia which is evident in every position and motion.

The diseases in which the staggering gait may occur are:

1. Acute alcoholism.
2. Multiple sclerosis.
3. Cerebellar disease.
4. Brain tumors.
5. Multiple neuritis.
6. General paresis.

9. **Hysteric Gaits.**—The hysteric gait may be of three types, according as to what part is involved in the functional paralysis. There may be a monoplegia, a hemiplegia, or a paraplegia; the gait will of necessity vary in each instance. If a monoplegia, as a rule the foot will be more affected than the leg; if a hemiplegia, the facial muscles and those of the trunk usually escape; if a paraplegia, there are almost invariably contractures and marked incapacity. The characteristics of the hysteric gaits are sudden development, complete paralysis, and preservation of automatic movements. There may be contractures or the paralysis may be flaccid. There is no marked rigidity, and under sudden stress the patient may invol-

untarily use the paralyzed limb. In the monoplegic the foot does not leave the ground: it is dragged; in the hemiplegic the leg is dragged, the sole again not leaving the ground; in the paraplegic, it being manifestly impossible for the patient to drag both legs successfully, resort is had to a crutch. The type which is most apt to develop contractures is the paraplegic. Oppenheim describes the gait as that of a child walking on stilts. In the hysteric paralyses there are apt to be, associated with the paralysis, stigmata and marks of degeneration.

The gait of hysteric hemiplegia differs from that of organic hemiplegia in that it lacks the characteristic deformity, the typical side-wheel motion, and in that it presents a more completely paralyzed limb. Usually the leg hangs like a flail, and either is completely raised from the ground or else the sole never leaves the floor. If the latter, the patient drags the leg behind as though held by ball and chain.

The unaffected leg is perfectly normal. In this gait therefore there can hardly be said to exist an abnormality of step. The body as a rule leans toward the affected side.

The principal characteristics of the hysteric gaits are:

1. Complete palsy.
2. Drag-foot.
3. Ability to use the limb in emergency.

These gaits occur only in hysteric paralyses.

Astasia-abasia is really one of the forms of hysteria. The leading characteristic in this condition is inability of the patient to stand or walk, while yet possessing in every other movement perfect control of the legs. In bed, in the sitting posture, even in the position of crawling, the patient is able to make perfect use of the legs. When,

however, he begins to walk, there results complete failure. The first few steps may be taken in the normal way, then they become slower, and in a very few moments cease altogether. The patient's expression is one of anxious distress and fear. He looks about for help, rubs his legs, tries to lift them, and finally in sheer desperation and discouragement gives up and drops to the floor. The condition is rare.

10. **The Gait of Arteriosclerosis.**—There is still one more gait different from all the others, yet not particularly distinctive in character. This is the gait of arteriosclerosis, sometimes called the senile gait. The patient complains of difficulty in walking, yet without being able to define exactly what is the obstacle. The legs are weak, the steps irregular, but not necessarily ataxic. There is unexpectedly a halting. The patient feels unable to advance. Each step appears to increase the difficulty. Or, again, the halting may be absent and the patient may be able to advance, yet the legs simulate a scissors-like movement, or the feet so interfere one with the other as to render progress practically impossible. There is no gait in which the patient is more conscious of his infirmity and more sensible of his disability than in the gait of arteriosclerosis.

The attitude which accompanies this gait is one of old age, with its attendant slowness and rigidity. The blood-pressure is high, the circulation weak, and there is every evidence of beginning senility. It is always well to remember that this gait is rarely seen in a man under fifty; in other words, the gait of arteriosclerosis, like the gait of festination, is generally an accompaniment of gray hairs. The reverse is true of the ataxic gait.

11. **The Normal Gait.**—The normal gait has been sufficiently described at the beginning of the chapter.

In addition to these gaits, typical and classic as they are, there are others that, although not particularly individual, and not specially diagnostic in any condition, are yet distinct enough to warrant a description.

There is the gait which occurs in the varied and progressive stages of *paresis*. Frequently in some of the protean diseases it is not possible to apply to it any particular train of symptoms. In paresis there may be an ataxic, a steppage, a spastic, or a hemiplegic gait. Yet it should be borne in mind that in paresis there is an attitude and manner of walking which occurs with sufficient frequency in the incipient and more advanced cases to warrant at least our describing it and putting it under the head of the paretic gait.

In the beginning of paresis, while there is no characteristic movement of the legs, there is evidence of a general weakness. This results in an incomplete adjustment of the muscular movements, together with a marked slowness and shortened step. As a result of the weakness of the legs, there is a corresponding bending at the knees, which gives to the attitude that of a beginning senility. The effort to progress is great, and the patient evidently labors. Together with this is a loss of tone of the facial muscles, a lack of expression, and an appearance of beginning dementia. As the disease advances the gait becomes more pronounced. The patient requires greater efforts to progress. The speed is much slower, the general weakness more apparent, and the evidences of dementia more pronounced.

It should never be forgotten that in paresis the dis-

ease may affect only certain groups of muscles, so that in this way a paretic may present a drag-foot or a hemiplegic gait. He may also show a typical ataxic or a typical spastic gait.

Hereditary chorea is a condition which presents a gait apart. The peculiarity varies with the progress of the disease. When the condition is well pronounced, the entire body is constantly in motion. The legs, feet, and body move from side to side and place to place. Oppenheim describes it by saying that the movement is rocking, with flinging movements of the arms and scraping movements of the legs. Again, the patient may suddenly come to a momentary standstill. The word which best describes the motion is the word "clam-like."

Sciatica may at times be diagnosed from its gait. In this condition the lameness is limited to one leg. The muscles supplied by the sciatic nerve are idle. The lower part of the leg is kept in a position of slight flexion; the thigh is abducted. Any effort to extend the leg is avoided. The patient gives the impression that the disabled leg is painful, and uses it as little as possible. The foot is raised at the heel, and the patient, if he progresses at all, does so on the ball of the big toe. There may be a slight bending of the body toward the affected side. There are always great deliberation and caution attendant on every effort to progress. The facies are those of a patient in pain.

Mixed gaits often occur. These are the result of the situation of the pathologic lesion. There may be a spastic-ataxic, or clumsy-steppage gait, or, in fact, any combination may exist. It is also possible to have two diseases occur in the same individual, so that a mixture of festi-

nation and hemiplegia, or steppage and festination, may be present.

Again, certain factors may be present which serve either to modify or confuse the gait. Tremor is very marked and, as sometimes happens in hemiplegia or paralysis agitans, will completely alter the appearance of the picture. Pain, resulting either from trauma, neoplasm, or hysteria, will also behave in the same way.

Finally, it should always be borne in mind that it is possible for a patient to have one type of gait occurring in one leg, with a wholly different one in the other; thus, a patient may have a hemiplegia on the right side, and at the same time a drop-foot on the left.

CHAPTER VIII

ATAXIA

Ataxia, derived from a Greek word meaning disorder, is the name applied to a condition that occurs in many diseases, and that may result from a lesion in various parts of the nervous system.

Man executes muscular movements by bringing into play varied groups of muscles. These he controls and moves in a definite, regular, systematic manner. These movements emanate from centers, situated in various parts of the central nervous system. A disturbance of these centers, therefore, will cause these muscular movements to be indefinite, irregular, and lacking in system. Such a disturbance we call ataxia. Again, the centers which control these movements, themselves normal, may yet fail to receive the necessary stimulation from the extremities, and in this way the resulting movements may be irregular and ataxic, or finally, as Leyden has pointed out, the joints may be so anesthetic as to preclude the sending of the proper impulse to the center, and in this way the resulting movements may be ataxic. It is, therefore, evident that not only may a lesion in the brain centers disturb this coördination, but also one that may be situated in the afferent tracts running to the brain centers. Thus, the lesion may be in the cerebrum, in the cerebellum, or in any part of the afferent tracts, either in the posterior nerve-roots themselves or in the posterior columns, or in the peripheral nerves.

Ataxia must not be confused with paralysis. It does not necessarily mean loss of power, but rather misapplied power. Ataxia must not be confused with anesthesia; it may be present with or without anesthesia. In one instance it may occur as the result of an anesthetic joint, because in this case the impulse to the center is transmitted either erroneously or not at all; it may, on the other hand, occur as the result of a central lesion with no anesthesia or with anesthesia which is secondary to some wholly different condition.

Ataxia must not be confused with tremor. In tremor the disorder of motion is regular, persistent, and more or less constant; it is a change in the number of oscillations which pass down the muscle. In ataxia the disorder of motion is more irregular, more jerky, and less persistent. It is due to faulty management and control of muscles. In tremor the muscle trembles, but the patient can control it; in ataxia there is no trembling, but the patient fails to gage his movements. Either he falls short or overshoots the goal.

Ataxia and incoördination differ only in degree. In other words, ataxia is advanced incoördination; incoördination is mild ataxia. The varieties and causes of ataxia are:

I. As to nature:
   1. Motor.
   2. Static.
   3. Cerebellar.

II. As to distribution:
   1. Hemi-ataxia.
   2. Both arms or both legs.
   3. All extremities.

III. Causes of ataxia:
1. Lesions affecting deep sensibility, as in tabes.
2. Toxic lesions of peripheral nerves, as in peripheral neuritis.
3. Lesions in higher centers. Brain—tumors in frontal or parietal lobe, inner capsule, and corpus quadrigeminus. Cerebellum—hemorrhage, softening, and abscess.
4. Lesions of pons, medulla, and cord, as hemorrhage, sclerosis, etc.
5. Congenital defects.

IV. The important ataxias:
1. Locomotor ataxia.
2. Cerebellar ataxia.
3. Disseminated sclerosis.
4. Acute disseminated myelitis.
5. Friedreich's ataxia.
6. Ataxic paraplegia.
7. Hysteric ataxia.
8. Miscellaneous.

As to Nature

1. **Motor Ataxia.**—The word motor when applied to ataxia occupies the position of a qualifying adjective. It is the form of ataxia which appears upon motion; at rest it is absent. The distribution may be local or general. Distribution in no way affects it.

2. **Static ataxia** is failure to coördinate while at rest. It includes the inability to hold the outstretched arm still and the inability to stand upright without swaying. Static ataxia occurs with aggravated forms of motor ataxia. A patient who suffers from ataxia when standing

is almost sure to suffer from it while in motion, whereas, on the other hand, a patient who suffers from motor ataxia does not necessarily suffer from static ataxia.

3. **Cerebellar ataxia** is the type which results from disease of the cerebellum. Another name for it is the

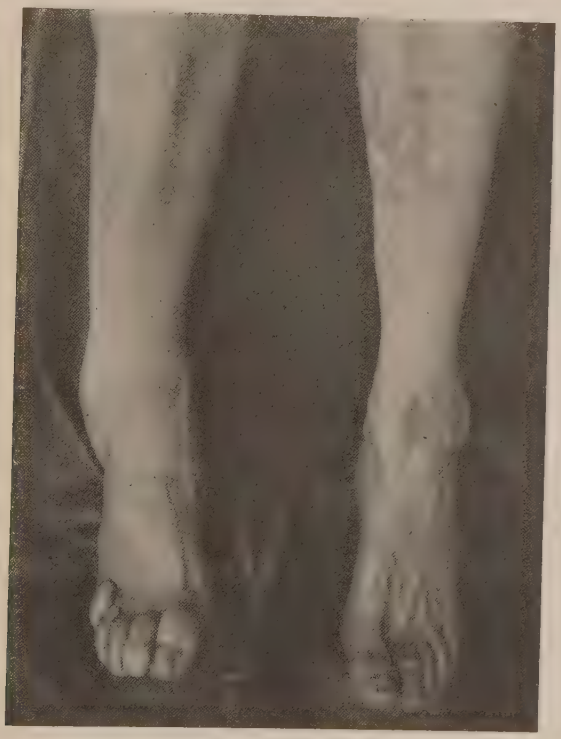


Fig. 40.—Charcot ankle in tabes.

term applied to it by Babinski, "cerebellar asynergia." It is one of the most important forms and one that the student would do well to familiarize himself with thoroughly. It is preëminently a disturbance in motion, and

differs from any other type of ataxia. It lacks disturbance of sensation. The resulting incoördination is due to three factors: first, the presence of vertigo; second, the existence of hypotonia; third, the lack of coördination between cerebellum and cortex.

Cerebellar ataxia may affect all four extremities; its most common distribution, however, is the legs. Therefore, the best illustration of this form of ataxia is seen in the gait, which has been described elsewhere. The difference between this form of ataxia and that which occurs in tabes is that, in the latter, there is some tendency to excessive movement, as the result of interference with the sensory impulses, while in the former there is a tendency to uncertain movement, as a result of a lesion in the coördinating center. In one the movement is excessive; in the other it is uncertain.

Fig. 41.—Charcot knee.

The particular lesions of the cerebellum which cause cerebellar ataxia are:

1. Tumor.
2. Abscess.
3. Maldevelopment.
4. Sclerosis.

5. Inflammation of the cerebellum.
6. Softening of the cerebellum.

Diseases in which the Gait of Cerebellar Ataxia Occurs

1. Early stages of myelitis.
2. Diphtheritic paralysis.
3. Disseminated sclerosis.
4. Friedreich's ataxia.
5. Diseases of the quadrate bodies and frontal lobes.
6. Diseases of the cerebellar cortex, crura cerebri.
7. Bulbar softening.

Romberg's sign is the name which is given to the test for ataxia. It derived its name from the man who first introduced it. To elicit the Romberg sign have the patient stand in soldier fashion, that is, with the hands to the side and the feet so close together that both toes and heels touch and with the eyes closed. Then stand some little distance in front of him. Observe carefully whether he stands straight or whether he sways. The normal man should and can stand thus in a steady manner without swaying. If he sways, the Romberg symptom is present. In other words, the presence of the Romberg sign is an indication of some disturbance in equilibrium.

Diseases in which the Romberg Symptom Occurs

1. Locomotor ataxia.
2. General paresis.
3. Multiple sclerosis.
4. Multiple neuritis.
5. Brain tumor.
6. Cerebrospinal syphilis.

As to Distribution

The distribution of ataxia may be general or local; it may involve the upper or lower extremities, or all four. It may or may not involve the head.

Hemi-ataxia describes a condition in which one-half of the body is ataxic. The occurrence of this condition is unusual; its principal importance lies in the fact that it is found with lesions of the optic thalamus.

Causes

The causes of ataxia are numerous.

1. It may result from lesions affecting deep sensibility. Any interference with the deep sensations, those of weight, movement, position, etc., will result in faulty transmission of the impulses running to the center. In other words, there will be an interference with the afferent tracts. The inevitable result of such a state of affairs will be defective coördination or ataxia. This is best illustrated in the ataxia of tabes.

2. It may result from toxic lesions of peripheral nerves, as in peripheral neuritis. Here there is a disturbance of the more superficial sensibility. Anesthesia of the legs, feet, arms, and hands takes place; there is the same interference with the transmission of afferent impulses, and again ataxia results.

3. It may result from lesions in the higher centers. The center of equilibrium is situated in the brain. Any lesion in either the cerebellum or in certain portions of the cerebrum will interfere with the centers of coördination and ataxia will result. Such lesions may be tumors in the frontal or parietal lobe, the inner capsule, or corpus quadrigeminus. The lesions in the pons, medulla, and cord

which may result in ataxia are sclerosis, abscess, hemorrhage, etc.

To summarize then: The causes of ataxia can really be placed under two headings—one an interference with the afferent tracts running to the centers of equilibrium, the other a lesion affecting the centers themselves. The two types of ataxia vary somewhat—the best illustration of the first is that seen in tabes, of the second that seen in lesions of the cerebellum.

To examine for ataxia it is best to adopt the following systematic method. Examine:

1. The arms.
2. The legs.
3. The trunk and head muscles.
4. Speech, tongue, and handwriting.

To **examine for ataxia in the arms** have the patient hold the arms at right angles, with all the fingers flexed except the index; then, while the eyes are shut, tell him to touch the tip of the nose with each index-finger in turn. The normal man can do this without tremor. A slight degree of ataxia will cause a blunder; a marked degree of ataxia will result in absolute failure. After this test make the patient try to approximate the finger-tips of the two index-fingers, first with the eyes open, and then with them shut. Next have him touch the finger of the examiner as the latter approaches with extended hand. Finally, have him try to pick up objects, and button and unbutton his clothes. Ataxia will render his efforts ungainly and vain; a very ataxic patient cannot button and unbutton his clothes.

To **examine for ataxia in the legs** make the patient walk, follow a pattern in the carpet, and heel and toe a

line. Have him try this latter motion with eyes shut. Next essay the Romberg test. Have him stand with both feet together, heels and toes touching, eyes shut, and arms to the side. This will test the sense of equilibrium. The normal man is able to stand for a few seconds in this position without swaying. The significance of the Romberg test is a disturbance of coördination; it occurs in locomotor ataxia, general paresis, and multiple sclerosis, some forms of multiple neuritis, and brain tumor. Another method of testing for ataxia of the legs is as follows: Have the patient in bed. Ask him to try to touch the foot, the ankle, and the knee of one leg with the heel of the other. The importance of this is twofold. It elicits slight degrees of ataxia, and it also enables us to differentiate between static ataxia and that of the cerebellar type.

To test for ataxia of the head and trunk muscles tell the patient to approximate the head to certain fixed objects.

Varieties of Ataxia

The important ataxias are:

1. **Locomotor Ataxia, or Tabes.**—Here there is an interference with deep sensibility. The defect lies in the transmission of the afferent impulse to the center. The center itself is intact. The type of ataxia resulting may be motor or static according as the condition is advanced or not. It can never be cerebellar.

The cardinal signs of tabes are the Argyll Robertson pupils, the lightning pains, the absent knee-jerks, the ataxic gait, and the crises.

Tabes in women is unusual, but does occur. Its one peculiar characteristic is that female tabetics are less apt to be ataxic than males. This is probably due to the fact

that the skirts of women prevent their looking at the ataxic movements of the feet. There is no doubt that a tabetic walks better and is less ataxic when he dispenses with the use of his eyes; one of the first lessons in Fraenkel's movements is for the patient to avert the eyes from the legs. It is a fact that a blind tabetic is rarely ataxic, and that an ataxic tabetic usually has the use of his eyes. A student will often notice in the wards of a neurologic hospital that the tabetic with the use of his eyes will have much more occasion to use a stick than the tabetic who has lost the use of his eyes. Those tabetics who are free from crises of pain are apt to be afflicted with gastric crises, and vice versâ.

Fig. 42.—Arrows indicate area and type of pain in locomotor ataxia.

Juvenile tabes is a term applied to tabes occurring in the very young. Though unusual, there are instances on record in which it has developed in the teens.

2. **Cerebellar Ataxia.**—Here the interference is in the center of equilibrium. There is no defect in the transmission of the afferent impulse. The type of ataxia is somewhat different. In walking, the body appears to run away from the legs. Again, in this form the patient is very apt to lean or walk to one particular side. This is due to the fact that the lesion may be situated in various parts of the cerebellum. Usually the leaning is to the side of the lesion. Not only does

he walk and lean to the side of the lesion, but he turns persistently toward that side. In cerebellar ataxia there is little control.

3. **Disseminated Sclerosis.**—Here the interference may be of either type, according to where the patch of sclerotic tissue lies. The very meaning of the word disseminate intimates that the lesion may be scattered.

4. **Acute disseminated myelitis** occupies very much the same position.

5. **Friedreich's ataxia,** or *hereditary ataxia,* is a congenital condition, developing early in life, and is due to a maldevelopment of certain portions of the spinal cord, particularly the posterior and lateral columns. Remember that in this condition the center of coördination is itself spared, whereas the pathologic lesion lies in the tract running to the center—the afferent tract. There are no sensory disturbances, and the ataxia is increased by closing the eyes.

The principal symptoms of Friedreich's ataxia are:

1. Incoördination.
2. Loss of knee-jerks.
3. Nystagmus.
4. Thickened speech.
5. Lateral curvature of the spine.
6. Deformity of the feet.

A practical point to remember is that in Friedreich's ataxia the plantar reflex is of the extensor type, whereas in locomotor ataxia the plantar reflex is of the flexor type.

Marie's *hereditary cerebellar ataxia* is due to a lesion of the cerebellum itself. It is difficult to differentiate it from Friedreich's ataxia, as the two are much alike. It is perhaps better to say that the two conditions belong to the same disease. In one the cerebellar type predom-

inates; in the other, the spinal. In Maric's ataxia the reflexes are not lost.

6. **Ataxic Paraplegia.**—Here the disturbance is purely one of the afferent tracts. The cardinal signs of this disease are ataxia, exaggerated knee-jerks, and a steadily increasing paralysis of the lower extremities.

7. **Hysteria.**

Hysteric Ataxia.—A form of ataxia may occur in hysteria, which, like all hysteric conditions, is more apt to resemble the same condition in other diseases. In this particular instance it resembles the ataxia that is found in multiple sclerosis. It is a form of ataxia which is not difficult to recognize, because it is performed in a more purposeless way, and displays movements which are de-

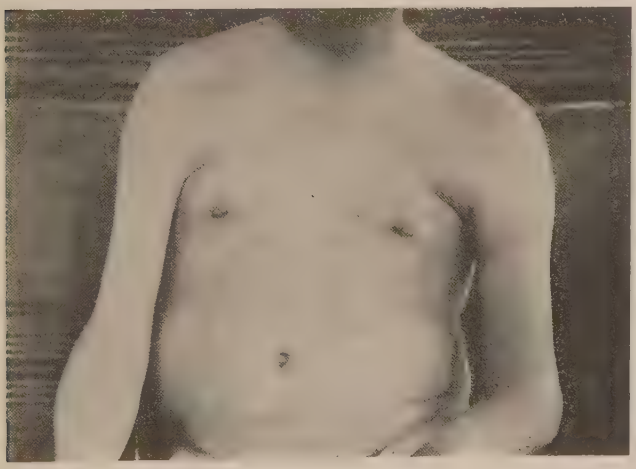


Fig. 43.—Atrophy of arm.

cidedly excessive. This latter point is best brought out by Oppenheim, who speaks of a patient that, when told to touch his nose with his finger, not only failed to

come near his nose, but went to so great a distance as to touch his eye. Another, when diverted and made to look at the examiner, was able to dispense with the ataxic movements, and appear quiet and normal.

8. **Miscellaneous Ataxias.**—In many instances the use of the word ataxia is arbitrary; in others it describes a particular condition.

The following list includes some of the miscellaneous types of ataxia: bulbar, diphtheritic, drunkard's, hereditary, hereditary cerebellar, locomotor, spinal, Briquet's, hemi- and posthemi-plegic, toxic, fatigue.

Bulbar Ataxia.—Of this form Oppenheim says: "Bulbar ataxia has the character of so-called sensory, cerebellar, and motor ataxia, or may show a combination of these types."

This form of ataxia is really a difficulty in articulation —a difficulty in performing the necessary muscular movements to express language. It may be a nuclear lesion, or may be due to a paralysis of the organs of speech.

Diphtheric Ataxia.—This is general in character, and is a form which follows an attack of diphtheria and accompanies the paralysis. It involves the hands and legs, and is very rapid in onset.

Drunkard's or *toxic ataxia* is the result of acute alcoholism. It is due to an affection of the higher centers, and shows both in the patient's gait and speech. It is, therefore, both cerebellar and cerebral.

Fatigue Ataxia.—Fatigue ataxia is best elicited in the incoördination, trembling, and uncertainty that accompany some of the occupation neuroses.

In the ataxic type of *multiple neuritis* Dresgfeld distinguishes two varieties—the paralytic and the ataxic. In the ataxic he groups those cases in which the muscular

sense was so greatly impaired as to bring about a condition of ataxia. This is the type which is so easily confused with tabes: the type to which the name of pseudotabes has been given. Starr calls attention to this fact, that ataxia is so exclusively limited to this type that it may occur coincident with paralysis.

The same division can be made of arsenical neuritis.

Toxic paraplegia is the name given to a disease the symptoms of which are weakness and rigidity of the lower limbs, together with exaggerated reflexes and ataxia.

Spinal Diseases in which Ataxia Occurs

1. Locomotor ataxia (tabes dorsalis).
2. Friedreich's disease (hereditary ataxia).
3. Ataxic paraplegia.
4. Combined posterolateral degeneration associated with anemia and various toxic conditions.
5. Some form of spinal syphilis (Erb's spinal syphilitic paraplegia).
6. Occasionally disseminated sclerosis.
7. Myelitis—disseminated, acute, and of infectious origin.

Other diseases causing ataxia in which the primary lesion is *not* in the spinal cord:

1. Tumors and other lesions of the cerebellum.
2. Hereditary cerebellar ataxia (of Marie).
3. The acute ataxia of Leyden (due to disseminated inflammatory patches in medulla, pons, and crura).
4. Occasionally tumors of the prefrontal region.
5. Internal ear disease.
6. Rare forms of peripheral neuritis (ataxic forms).
7. Occasionally hysteria.

CHAPTER IX

CONVULSIONS

A CONVULSION is an unnatural, violent, irregular contraction of the muscular parts.

In studying convulsions inspection will yield the greatest rewards. The student must observe with great care and intelligence the premonitory signs, the presence or absence of an aura, the onset, the course of the convulsion, the duration, and the distribution. He must note whether or not an outcry is present, the condition and position of the eyes, tongue, mouth, and sphincters. He should take the pulse and respiration, observe whether or not the patient passes water during or after the convulsive stage, and, finally, take into account the postparoxysmal condition and the temperature.

Convulsive movements are so frequent and so similar in appearance that a differential diagnosis can be made only after collecting and weighing much data. The epileptiform type is the most usual and therefore the one with which the student must be most familiar. It is the one to suspect in every case of convulsion.

The best arrangement and classification of convulsions is the following:

CLASSIFICATION OF CONVULSIONS

I. EPILEPSY:

- (*a*) Grand mal.
- (*b*) Petit mal.
- (*c*) General.
- (*d*) Jacksonian.

II. Convulsions due to Pressure:

(*a*) Brain tumor—general or localized.
(*b*) Brain abscess—general or localized.
(*c*) Bony pressure { 1. Tumor. 2. Depressed fracture.
(*d*) Hydrocephalus—general.

III. Vascular Diseases:

(*a*) Hemorrhage during apoplexy.
(*b*) Thrombosis { 1. Cerebral. 2. Sinus.
(*c*) Syphilitic arteritis { 1. Thrombosis. 2. Hemorrhage. 3. Aneurysm.
(*d*) Meningeal hemorrhage.
(*e*) Anemia of brain.
(*f*) Hyperemia of brain.

IV. Toxic Action:

(*a*) Drugs, as strychnin, lead, alcohol, arsenic, morphin, prussic acid, atropin, aconite.
(*b*) Uremia.
(*c*) Eclampsia.
(*d*) Tetany.

V. Infection:

(*a*) Syphilis { 1. Diffuse. 2. Gummatous.
(*b*) Meningitis { 1. Cerebrospinal. 2. Tuberculous. 3. Serous. 4. Pachymeningitis.
(*c*) General paresis.
(*d*) Tetanus.
(*e*) Parasitic invasion of *Cysticercus cellulosæ*.

VI. Functional:

Hysteria.

I. **Epilepsy.**—(*a*) *The Grand Mal.*—The grand mal is a general convulsion which is severe and violent. It is preceded by an aura, accompanied by an outcry, and followed by a complete loss of consciousness. The aura may assume one of many types. It is immediately followed by a twitching, which quickly becomes general and violent. The patient is cyanosed, froths at the mouth, rolls the head to one side, and cries out. He falls to the floor, bites his tongue, passes his water, breathes in a stertorous way, and after two or three minutes lapses into a deep sleep.

(*b*) *The Petit Mal of Epilepsy.*—The petit mal is really no convulsion at all. It may or may not be preceded by an aura, is not always accompanied by a loss of consciousness, and never by an outcry. It may simply take the form of dizziness, loss of memory, or absent-mindedness.

(*c*) *General.*—The general convulsion is so called to distinguish it from the Jacksonian, and is in reality grand mal.

(*d*) *Jacksonian Convulsion.*—The Jacksonian convulsion is localized epilepsy. In this form the convulsion begins in a particular muscle or group of muscles, and may or may not extend to a general involvement. If it does extend, it is in a definite and systematic manner. The cause of the Jacksonian convulsion is a focus of irritation in that particular part of the cortex of the brain which controls the particular group of muscles.

II. **Convulsions Due to Pressure.**—(*a*) The general convulsion occurring in *tumors of the brain* is violent and apoplectiform. It is the result of intracranial irritation or pressure.

The second, or localized, type is really a Jacksonian

convulsion, that is to say, it involves one group of muscles or one extremity. Like the Jacksonian convulsion, it may begin locally, then spread and become general. This type should be observed with care and precision; it is of importance in aiding the diagnosis, as it serves to localize the cause of irritation. It should be kept in mind that some parts of the cortex are more sensitive than others, and therefore much more apt to discharge.

In tumors of the cerebellum convulsions do occur, but with less frequency.

(*b*) *In abscess* convulsions are rare; they are most apt to appear just before death or when the encephalitis begins to break down.

(*c*) *Bony pressure*, either from a spicula of bone, new-growth, or depressed fracture, behaves in the same manner as does a neoplasm and causes the same kind of convulsion.

(*d*) *In hydrocephalus* the convulsion is a late manifestation.

III. **Vascular Diseases.**—*Hemorrhage during an apoplectic stroke* may be initiated, accompanied, or followed by convulsions. They are, however, unusual and, unless the hemorrhage is severe, do not occur. Their presence indicates that the bleeding has become extensive and probably has invaded the ventricles. These convulsions may be general, local, or unilateral. If unilateral, Starr says they are an infallible sign of a lesion of the cortex in the central region. In the case of children suffering from cerebral hemorrhage convulsions are still more usual and frequent. The type of convulsion occurring in cerebral hemorrhage is, of course, apoplectiform.

In *sinus thrombosis* convulsions occur. They are either general or unilateral, and are more frequent in children than in adults.

In *meningitis*, especially in the meningitis of childhood, convulsions are frequent. They occur early, are violent, and persist. They may be general, unilateral, or they may assume the form of a localized spasmodic twitching. They are most frequent in tubercular meningitis, in which disease they are general in character; in the epidemic type of meningitis they occur with almost equal frequency.

In *syphilis of the brain* the type of convulsion is epileptiform and general.

In *cerebral anemia* general convulsions occur, provided the loss of blood is severe. They are by no means infrequent, and are general in character.

In *cerebral hyperemia* convulsions are fairly frequent. They occur in the advanced stage, are preceded by mental irritability, confusion, vertigo, apathy, and flashes of light, and accompanied by a rise in the blood-pressure. They are general in character, violent, and usually several closely follow one another.

IV. **Toxic convulsions** follow overdoses of strychnin, alcohol, lead, aconite, atropin, arsenic, morphin, and prussic acid. They are most frequent in strychnin poisoning, less so in alcohol, and rather unusual in lead. They are general, violent, and epileptiform. In strychnin poisoning the convulsions present both clonic and tonic spasms, follow each other frequently, and finally attain the stage of opisthotonos. In this condition the patient may reach so great a state of susceptibility that the slightest irritation, such as the presence of a light

or the rustling of a newspaper, may induce a series of these convulsions..

V. **Infection.**—*Convulsions in general paresis*, as a rule, usher in and accompany the third stage, although they may occur early in the disease. They assume varied forms: from the simple congestive attack, which occurs in incipient paresis, to the violent general convulsion of the terminal stages. These latter may be apoplectiform, epileptiform, or general.

In *tetanus* convulsions begin as spasms in the fingers and toes and gradually extend toward the trunk until eventually they involve the entire body. Simultaneously with this progressive involvement the spasm becomes a convulsion, which steadily increases, in severity until there results a general opisthotonos.

VI. **The hysteric convulsion** is one which occurs in all the different types of hysteria, in the minor, the major, and in catalepsy. Pitres says that in hysteria 82 per cent. of the women develop the convulsive type, whereas only 22 per cent. of the men.

The principal feature of this type of convulsion is that the period of motor activity is preceded by a period of preparation, and followed by one of mental excitement or delirium. The convulsion itself may be one of two kinds, that is, either clownish or emotional. The attack, if of the latter type, may assume tremendous dimensions and cause the onlookers the greatest discomfort and even fear. The patient may assume the opisthotonos position. The preparatory period is one in which the patient may be depressed or irritable and may display a good deal of dissatisfaction with surroundings. There is also much motor activity.

CHAPTER X

SENSATION

The best, the most comprehensive, and the one plan most widely adopted for explaining the sensory disturbances is that suggested by Drs. Henry Head and James Sherren.

Their theory is that afferent impulses coming from the periphery are conducted along distinct classes of nerve-fibers. These three classes are named deep, intermediate, and superficial sensibility. This plan extends to the spinal roots. So soon as these impulses enter the spinal posterior roots nature's arrangement is changed. There is a complete rearrangement. There is no longer any deep, intermediate, or superficial sensibility. There are tracts; in each tract are gathered together, so to speak, all the fibers of sensation of that particular type, regardless of whether they are superficial, deep, or intermediate. In the spinal cord there are the tracts of pain, temperature, and touch.

The Head plan divides the sensory mechanism into two parts:

I. The sensory peripheral mechanism.

II. The sensory spinal mechanism.

In the sensory peripheral mechanism are three sets of impulses, which travel by different paths.

1. *Those Which Affect Deep Sensibility.*—In this type the impulses are produced by pressure. Thus is

recognized the sensation of muscular and articular movement.

2. *Those Which Affect Intermediate Sensibility.*—In this type the impulses are produced by painful cutaneous stimuli and the extremes of temperature. This type

Fig. 44.—Syringomyelia, showing the winged or angel scapula, the scoliosis, and the atrophy of the arm.

has been named by Head the protopathic. This intermediate type is more wide-spread in its distribution than the others and is accompanied by tingling.

3. *Those Which Affect Superficial Sensibility.*—In this

type the impulses are produced by touch in its restricted meaning, that is, the impulses are produced by gentle touches, minor degrees of temperature, and the recognition of two points of a compass as two points. This type has been named the epicritic.

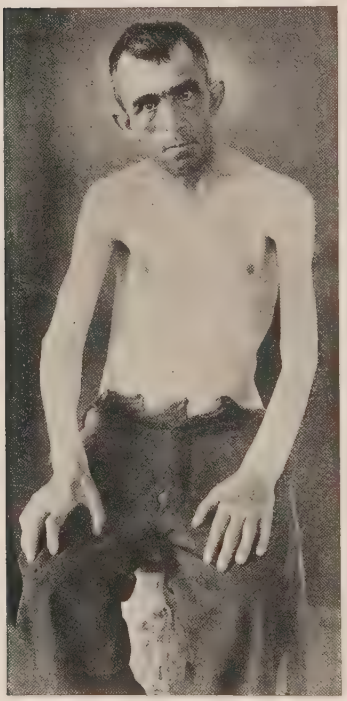


Fig. 45.—Syringomyelia, showing atrophy of the hands and arms, right claw-hand, lateral scoliosis, and sympathetic paralysis on the left.

The unit of the protopathic supply is the posterior root.

The unit of the epicritic supply is the peripheral nerve.

Sherren says division of *posterior roots* produces an area of loss of protopathic greater than the area of loss of light touch.

Division of *peripheral nerves* produces an area of loss of protopathic either smaller or almost as extensive as the area of loss of light touch.

Sensory Spinal Mechanism. — In the spinal cord there is an entirely different mechanism. Here we have groups of sensations of:

Pain.
Temperature.
Touch.

Passive position and movement, together with discrimination of adjacent points.

The Method of Examination for Sensation

Many instruments and many methods have been devised to test sensation. The student will do best to discard all of the complicated ones, and rely on the simplest. The only instruments needed are a small camel's-hair brush, a pin, a compass, two test-tubes, one containing hot water, the other cold, and the examiner's fingers. The room should be quiet, the patient stripped and blindfolded. The student should then proceed in a systematic manner. Tell the patient to say "Yes" whenever he feels any sensation of touch, and then to qualify his statement by telling the location and nature of the sensation. Never say to a patient, "Do you feel *that?*" or "What was *that?*" as in some cases these questions give a clue as to the sensation. Do not forget to make the tests at irregular intervals and of a nature varying enough so that the patient will not know what test to expect.

(*a*) **Epicritic.**—First test for light sensibility; that is epicritic. Test for light touch, light pain, light differences in degrees of temperature, and the discrimination of distance between two points. The test for touch is made by touching, in the gentlest possible way, the skin with a camel's-hair brush, a piece of cotton-wool, or even with the fingers of the examiner. The test for pain is made by touching the skin with the point of a pin, the requirement of the patient being to tell whether it is the head or the point of the pin. The test of the degree of temperature is made by touching the skin with the two test-tubes, one of which contains water only a

trifle warmer than the body, and the other of which contains water only a trifle colder than the body. The discrimination test is made by a compass, or roughly by the two fingers of the examiner, the object being to determine the distance of the points at which the patient fails to appreciate the two points and feels the sensation of one point only. In this connection it is well to mention another delicate test. Move the camel's-hair brush or the pinpoint very lightly in either vertical or horizontal streaks on the skin, asking the patient to determine both the direction and distance traversed. This distance traversed includes really the compass test besides the sense of direction.

(*b*) **Protopathic.**—The protopathic sensibility is the name applied to more strenuous applications of the touch of the camel's-hair brush or finger, a rather quick jab of the pinpoint, and the difference between the test-tubes that are quite hot and those that are ice cold. Therefore, following the epicritic test, carry out the one for protopathic sensibility.

(*c*) **Deep Sensibility.**—Finally, examine for deep sensibility. This test involves the knowledge of the various positions of the arms, fingers, legs, and toes when the eyes are closed. This is done in several ways: 1. By asking the patient to bring his finger-tip to within an inch of his nose, and then stop without touching the nose. (This test will also demonstrate any tremors or incoordination, although not its primary object.) 2. Similarly, hold one hand or foot of the patient, asking him to touch it quickly with the other hand. When the deep sense is lost, the patient may reach in the wrong direction with the second hand. 3. Hold one extremity in an

awkward or unusual position, asking the patient to duplicate or imitate the position with the opposite limb. Sometimes it is of value to ask the patient to open the eyes and tell if the limb is where he thought it was. 4. Grasp the little finger or big toe between the thumb and forefinger. Move it up, down, and sideways, at the same time asking the patient to describe the direction of each movement. Bear in mind that it is necessary to grasp the lateral sides rather than the dorsal and palmar aspects, as the patient might tell from superficial sensation that the finger of the examiner touching the dorsal surface of the toe could do nothing else than flex. This is the most delicate of any of the tests for joint sense. It can be made even more delicate by moving the toe only slightly up or down instead of alternating between extreme flexion and extension. This sense as to passive motion is kinesthesia; its absence is called akinesthesia. 5. Test the recognition of weight by placing in the patient's hands coins or metallic articles of varying weights. 6. Test the stereognosis sense. This test involves size, shape, character of surface, and weight. It is the most complex and one of the most important of all the tests. To carry it out successfully the patient must have in his hands several articles of varying character, weight, and size. Without seeing them he must try to describe and distinguish them and their characteristics. The absence of this sense is called astereognosis and is described later on.

Sensory Disturbances

(*a*) Varieties as to distribution.
(*b*) Varieties as to nature.

(*a*) Varieties as to Distribution:

1. Over several muscle segments or several spinal nerve segments, as in tract disease of the cord.
2. Over individual segments, following trauma. As definite segments, as in ulnar nerve injury.
3. One-half the body, as in hysteria.
4. Both lower extremities, as in tumor of cord.
5. Generalized over the body, as in tabes.
6. Irregular distribution over various segments, not following the course of nerves, as in hysteria.

(*b*) Varieties as to Nature:

1. Touch.
   - (*a*) Hyperesthesia, excessive sensibility to touch and other stimuli.
   - (*b*) Hypesthesia, diminished sensibility to touch.
   - (*c*) Anesthesia, loss of tactile sensibility.
2. Pain.
   - (*a*) Hyperalgesia, excessive sensibility to pain.
   - (*b*) Hypalgesia, reduced sensibility to pain.
   - (*c*) Analgesia, loss of sensibility to pain.
3. Thermal.
   - (*a*) Increased.
   - (*b*) Decreased.
   - (*c*) Absent.
4. Dissociated anesthesia, in which tactile and pain sensibility is preserved, sensibility to heat and cold is diminished or lost.
5. Varieties of deep sensation.
   - (*a*) Kinesthesia, loss of sensation as to position of joints.
   - (*b*) Pallanesthesia, loss or diminution of the vibration sense.
   - (*c*) Astereognosis, loss of the power of recognition of objects by sense of touch.

Fig. 46.—Normal and atrophied hands.

Varieties and Occurrence of Disturbed Sensation

1. Thermal Anesthesia:
   - Syringomyelia.
   - Hysteria.
   - Tumor of cord.
2. Analgesia-hypalgesia:
   - Tabes.
   - Hysteria.
   - Tumor of cord.
3. Anesthesia-hypesthesia:
   - Tabes.
   - Trauma.
   - Myxedema.
   - Hysteria.
4. Dissociated Anesthesia:
   - Syringomyelia.
   - Hematomyelia.
   - Hysteria.
5. Kinesthesia:
   - Tabes.
6. Pallanesthesia:
   - Tabes.
7. Hyperalgesia:
   - Tumor of cord.
   - Hysteria.
   - Zoster.
8. Hyperesthesia:
   - Alcoholic multiple neuritis.
   - Hysteria.
   - Zoster.
9. Brown-Séquard Type:
   - Tumor of cord.

CHAPTER XI

REFLEXES

KNOWLEDGE of reflexes is essential to a knowledge of neurology. The student must know how to obtain reflexes, their relationship to symptoms, their significance, and the pathologic conditions which they foreshadow and declare. There are many reflexes, some of which are irrelevant to neurology. The student should learn to give close attention to the most important, and not to waste time over those of little value. First, it is best to understand just what a reflex is and what is reflex action. A reflex action, according to Webster, is "any action performed involuntarily, in consequence of an impulse or impression transmitted along afferent nerves to a nerve center, from which it is reflected to an efferent nerve, and so calls into action certain muscles, organs, or cells." In other words, a reflex action is the performance of a given action which results from a sensory impulse produced on the spinal cord, regardless of the patient's aid or knowledge. It is constantly seen in the acts of the lower animals.

To accomplish a reflex act it is necessary to have a complete and unimpaired mechanism. This mechanism consists of three parts: the sensory nerve running to the center, the center itself in the cord, and the motor nerve running from the center. These three parts together make up what is known as the reflex arc. Any

injury or interference with any portion of this arc will result in destroying the transmission of impulse and so bring about a loss of reflex. Thus, a stab wound, a patch of sclerotic tissue, or inflammation will break the arc, destroy the transmission of the impulse, and result in an absent reflex.

A certain degree of mental control is transmitted over all reflex acts through the spinal cord from the brain. If some injury or pathologic change takes place in the columns of the spinal cord, this cerebral control will be interfered with, the reflex will cease to be controlled, and there will result an exaggeration of the reflex. Again, if certain poisons are administered they will act as stimulants to the spinal cord, will irritate the centers, and so bring about an exaggeration of the reflex.

Thus there is one cause for a lost reflex act: a break in the reflex arc.

There are two causes for an exaggerated reflex act: removal of cerebral inhibition and overstimulation of the spinal centers.

In order to perform a reflex act properly, it is necessary that each of the three parts of the reflex arc should be intact. When they are intact, an impulse or stimulus given to the external skin or tendon is conveyed by the afferent fiber to the center, and by the center transmitted to the efferent fiber, which in turn stimulates the muscle or skin, and so produces the motor action. The integrity of the reflex, then, is wholly dependent upon the integrity of the reflex arc.

A break in the reflex arc will result in a lost reflex. Such a break may come from trauma, inflammation,

hemorrhage, a patch of sclerotic tissue, poison, degeneration, and new-growths.

A reflex arc, therefore, consists of three distinct parts:

1. The afferent fiber running to the center.
2. The efferent fiber running from the center.
3. The center, together with its connecting fibers, which serve as a link between the two.

Special Reflexes

There are four groups of reflexes:

I. Pupillary.
II. Superficial (skin and mucous membrane).
III. Deep (tendon).
IV. Organic.

I. Pupillary Reflexes

The pupillary reflexes consist of:

(*a*) The light reflex.
(*b*) The accommodation reflex.
(*c*) The ciliospinal reflex.

To test for the **light reflex,** examine each eye separately. The best way is to shade one eye and at the same time alternately admit light and darkness to the other. Normally, from the admission of light there should result a contraction of the pupil, and, from shading, a dilatation. Pathologically, the pupil may be either sluggish or absolutely immobile. The first condition is suggestive of a beginning immobile pupil. The second is a pupil which has become immobile.

To obtain the **accommodation reflex,** have the patient look first at a near and then at a distant object. Normally there should result a contraction and dilata-

tion. The loss of the power of accommodation is unusual; it may occur in certain lesions of the sympathetic system.

To obtain the **ciliospinal reflex,** pinch the skin of the neck. The result will be a dilatation of the pupil of the corresponding side. The spinal centers which contain the ciliospinal center are situated in the lowest cervical and highest dorsal regions.

II. The Superficial Reflexes

The superficial reflexes are not so important as the deep. They involve both the skin and mucous membrane. The method of obtaining them is by irritating the skin or mucous membrane, either by scratching or pinching. As a result of this stimulus, there follows a certain definite contraction. While it is admitted that the skin reflexes are controlled by certain centers in the cord, yet it is not admitted that the control is as thorough or as important as in the case of either the pupillary or the deep reflexes. There are many superficial reflexes. A list of the principal ones follows:

1. The corneal.
2. The conjunctival.
3. The ciliospinal.
4. The palatal.
5. The pharyngeal.
6. The scapulohumeral.
7. The epigastric.
8. The abdominal.
9. The cremasteric.
10. The plantar.
11. The Babinski.
12. The Oppenheim.
13. The Gordon.
14. The Chaddock.
15. The Schaefer.
16. The Bechterew-Mendel.
17. The palmar.
18. The sphincter.

The **corneal reflex** is produced by taking a very small piece of absorbent cotton, rolling it tightly, and carrying it over the conjunctiva to the cornea. Normally

the eye should wink. In the pathologic condition the patient will allow the examiner to place the cotton on the cornea without any movement on the part of the eyelid. At the same time the **conjunctival reflex** can be obtained. The centers which control these two reflexes

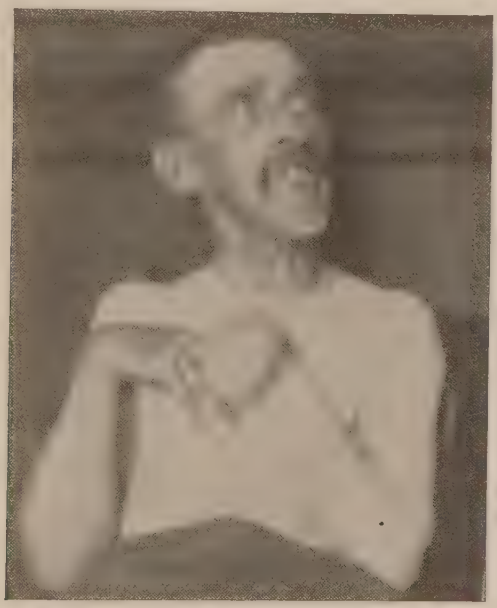


Fig. 47.—Athetosis duplex.

are not well defined. These reflexes are often absent in functional conditions. While their absence is very frequently an indication of hysteria, it should never be forgotten that they may be present even in mild cases of neurasthenia.

To obtain the **pharyngeal** or **palatal reflex** stroke the back of the throat with a spatula or some small wooden object. Coughing or gagging occurs normally, the right

side of the palate and pharynx moving simultaneously and equally to the left.

To obtain the **scapulohumeral reflex** stroke the skin over the scapula. There should result a contraction of the scapular muscles. In pyramidal tract lesions this reflex is exaggerated.

To obtain the **epigastric reflex** stroke the skin of the epigastrium. Normally there would result a contraction of the upper fibers of the recti muscles of the epigastric region. This reflex should be obtained independently on both sides. The segment of the spinal cord in which it is located is the seventh to the ninth dorsal.

The **abdominal reflex** is illustrated by irritation, in a similar manner, of the lower lateral part of the abdomen. Normally there should result a retraction of the abdominal muscles of the same side. The segment which controls this reflex is situated in the ninth to the twelfth dorsal. The absence of this reflex is not at all unusual in the crises of tabes and in multiple sclerosis. Following apoplexy the abdominal reflex is lost on the paralyzed side.

Diseases in which the Abdominal Reflex is Lost

1. Hemiplegia.
2. Multiple sclerosis.
3. Brain tumor.
4. Tabes (crises of).
5. Epidemic cerebrospinal meningitis.

The **cremasteric reflex** is obtained by scratching or pressing the inner side of the thigh. There results a retraction of the testicle of the same side. The segments in the spinal cord which control this reflex are the first and second lumbar.

The **plantar reflex** is, of all the cutaneous reflexes, the most important. To obtain it stimulate with some blunt object the sole of the foot. Have the patient elevate the leg, while the examiner, to place it in a passive position, supports the calf. Use either a blunt object or the thumb-nail of the examiner, slowly rubbed along the inner side of the sole of the foot toward the toe or from side to side. In the normal individual, as a result of such stimulation there would result a plantar flexion of the toes. This, of course, is best illustrated in the phenomena of tickling the sole of the foot. If, however, the reflex is abnormal, then there results a phenomenon which is known as the **Babinski.** This consists of a flexion of the smaller toes, a marked extension of the large toe, and sometimes a spreading in fan-like manner of the four smaller toes. This characteristic spreading of the toes has given to the reflex the name of the *signe de l'eventail.* The name of Babinski, by which it is commonly known, was given to it as a result of its first description by this French authority. The significance and importance of the Babinski reflex lies in the fact that its presence is indicative of an involvement of the pyramidal tracts. It is, therefore, a positive indication of involvement of the upper motor neuron type. It should never be forgotten that in infancy, up to the age of eighteen months, the pyramidal tracts remain undeveloped, and that, therefore, at that age the Babinski sign will be found, not pathologically, but physiologically. The Babinski reflex is a very important sign in the differential diagnosis between organic and functional conditions. It is well to remember that in hysteria the pure Babinski reflex is not present.

Diseases in which the Babinski Reflex Occurs

1. Multiple sclerosis.
2. Tumors of cord and brain.
3. Hemiplegia.
4. Spastic spinal paralysis.
5. Congenital spastic paralysis.
6. Friedreich's disease.
7. Meningitis.
8. Amyotrophic lateral sclerosis.
9. Fracture of spinal column.
10. Spinal caries.
11. Myelitis.
12. Poliomyelitis (occasionally).

Conditions in which the Plantar Reflex is of the Flexion Type

1. Tabes.
2. Peripheral neuritis.
3. Paralysis agitans.
4. Poliomyelitis.
5. Neurasthenia.
6. Chorea.

To obtain the **Oppenheim reflex** stroke from above downward the inner border of the tibia. The result is a contraction of the extensor muscles of the big toe.

To obtain **Gordon's reflex** make deep pressure on the muscles of the calf. There results an extension of the big toe.

To obtain the **Chaddock reflex** stimulate below the external malleolus. There results an extension of the hallux.

To obtain the **Schaefer reflex** pinch the Achilles. There results pain.

To obtain the **Bechterew-Mendel reflex** tap the toe tendons. There results an extension of the hallux. The first three sacral segments of the spinal cord control these six reflexes. These last six reflexes occur only in pyramidal tract lesions.

The **palmar reflex** is illustrated by stroking the palm of the hand, with the resultant flexion of the fingers and slight retraction of the hand. The normal thickness of the skin in this region will often make it impossible to illustrate this reflex. It is localized in the eighth cervical to the first dorsal.

The **sphincter reflex** is twofold—bladder and rectum. To obtain the sphincter reflex of the bladder introduce a sound. If present, the instrument will be gripped. To obtain the sphincter reflex of the rectum introduce a finger. Normally the sphincter contracts tightly over the finger.

Abnormalities of Sphincter Control:

1. Retention.
   - (*a*) Tabes.
   - (*b*) Tetany (sphincters may be tonically contracted).
2. Incontinence.

Varieties of Incontinence:

- (*a*) Continuously, as fast as any excretion is produced.
- (*b*) At intervals—whenever there is accumulation.
- (*c*) As result of distended bladder; as overflow.
- (*d*) Nocturnal enuresis.
- (*e*) Accidental, under emotions, or haste, or acute alcoholism.
- (*f*) Epileptic convulsion.

Occurrence of Incontinence:

(*a*) Tabes.
(*b*) Brain tumor.
(*c*) Tumor of the spinal cord.
(*d*) Multiple sclerosis.
(*e*) Idiocy.
(*f*) Epileptic convulsions.
(*g*) Senile dementia.
(*h*) Hysteria.
(*i*) Myelitis.
(*j*) Landry's paralysis.
(*k*) Cerebrospinal meningitis.
(*l*) Multiple neuritis (occasionally).

Deep Reflexes

The deep reflexes are many. The important ones are:

1. The jaw jerk.
2. The elbow, or triceps, jerk.
3. The wrist jerk.
4. The knee jerk.
5. The Achilles jerk.
6. Clonus—ankle, patella, leg, wrist, elbow.

To get the **jaw jerk** have the patient extend the chin, while at the same time the examiner places a flat substance, preferably an ivory paper-cutter, over the lower teeth. As a result of repeated tapping there occurs a contraction of the muscles which elevate the jaw. This is not a reflex of any very great importance, but, according to Beevor, it is frequent in amyotrophic lateral sclerosis.

To obtain the **triceps reflex** have the patient rest the arm on the examiner's hand with the forearm hanging

loosely. A slight tap, just above the elbow, at the insertion of the triceps muscle, ought then to cause an extension of the forearm. The segment of the spinal cord which controls the triceps jerk is the sixth cervical.

To obtain the **wrist jerk** support the flexed forearm while the hand of the patient is allowed to hang downward. A tap, either with a percussion hammer or with the fingers, will then bring forth a contraction of the hand. The blow should not be severe, and should be applied above the dorsal surface of the forearm in the radial end. The spinal segments which control the wrist jerk are the sixth to eighth cervical.

Diseases in which the Wrist Jerk may be Absent

1. Progressive muscular atrophy.
2. Erb's arm paralysis.
3. Any neuritis affecting the arms.
4. Poliomyelitis affecting the arms.
5. Tabes if the cervical cord is affected.
6. Syringomyelia if the cervical cord is affected.

Diseases in which the Wrist Jerk may be Exaggerated

Diseases affecting the upper motor neurons.

Amyotrophic lateral sclerosis.

The **knee jerk** is the most important of all reflexes. Unusual care and tact must be exercised to test for its presence. First have the patient cross one leg over the other. In this position feel for the lower end of the patella, and with a percussion hammer, a book, or the fingers strike the quadriceps tendon just below its insertion into the patella. Normally, there should occur an extension of the leg. Pathologically, there may occur

either an exaggeration of this phenomenon or a complete absence of movement on the part of the leg.

If the patient happens to be confined to his bed, each knee can be supported in the hand of the examiner. Neither of these methods, however, should be deemed satisfactory or final. The only proper way to test for the knee jerk is to have the patient sit on a table, with the legs hanging loosely, very much as though he were sitting on a country stile. If this method should fail the examiner ought not to be satisfied until he has tried additional methods, known as reinforcements. The Jendrassik method consists in having the patient clench his two hands, and, at the moment at which the examiner tests the knee jerk, pull the clenched hands in opposite directions. If this method should fail, the examiner should then try the method which goes by the name of Lauferauer's. This consists in having the patient sit with his soles flat on the floor, while he squeezes together his two hands. Just at this moment the patella tendon is tapped. If there is the slightest response, it will be evident to the examiner's other hand, which should hold the patient's quadriceps muscle. Still another method is to have the patient, while sitting on a table, read aloud rapidly, with a newspaper between him and his knees, the object being to convey to him the impression that his vision and speech are being tested. This method is probably the surest of all, and if under these conditions no reflex is obtained, it is safe to feel that the reflex is absent. In every instance where the presence of a knee-jerk is doubtful, the student must try each one of these methods. This holds true even in the case of bed patients. A bed patient can always be supported on

a table and induced either to clasp his hands or to read. It is surprising how many functional and hysteric cases that have been bedridden for years will under these

Fig. 48.—Atrophy resulting from alcoholic neuritis in a man formerly weighing over two hundred pounds.

conditions and none other present a reflex, thus enabling the examiner to diagnose a difficult case. Another name for the knee-jerk is the patellar reflex. Its absence is known as Westphal's sign. The spinal centers which

control it are situated in the second, third, and fourth lumbar nerve roots.

The knee jerk is absent in the following conditions:

1. In lesions of the afferent limb of the reflex arc.
2. In lesions of the efferent limb of the reflex arc.
3. In lesions of the gray matter at the level of the reflex arc.
4. In coma, exhaustion, narcosis, and spinal anesthesia.
5. In cerebral conditions in which there is increased pressure of the cerebrospinal fluid.
6. Congenital (rarely, but it does occur).
7. In pyrexial states.
8. In certain conditions of diseases of the cord above the reflex arc.
9. In clear transverse lesions of the cord.

Diseases in Which the Knee Jerk may be Absent

1. Tabes.
2. Neuritis (alcohol, diphtheria, diabetes, arsenic, grip).
3. Poliomyelitis.
4. Progressive muscular atrophy (Charcot-Marie Tooth type).
5. Coma preceding death.
6. Diabetes.
7. Syringomyelia.
8. Injuries to the crural nerve.
9. General paresis.
10. Beriberi.
11. Tumors of the pons.
12. Friedreich's disease.
13. Combined posterior and lateral column disease.

14. Muscular dystrophy.
15. Spinal meningitis.
16. Landry's paralysis.
17. Increased pressure of spinal fluid.
18. Cerebral meningitis.
19. Epidemic cerebrospinal meningitis.
20. Exophthalmic goiter (rarely).
21. Postepileptic conditions.

The knee jerk is exaggerated in the following conditions:

1. Any lesion of the pyramidal tracts.
2. Brain tumors.
3. Functional neuroses.
4. Toxic conditions (in the irritative stage).

Diseases in Which the Knee Jerk may be Exaggerated

1. Hemiplegia.
2. Multiple sclerosis.
3. Spastic paraplegia.
4. General paresis.
5. Brain tumor.
6. Tumor of the spinal cord.
7. Multiple neuritis in early stage.
8. Hysteria.
9. Spinal meningitis.
10. Myelitis.

Such a condition as inequality or irregularity of the knee jerks may occur, so that the knee jerk on one side may be present while the corresponding one is absent. Or, it may be exaggerated, with the other either absent or normal. This state of affairs may follow apoplexy, where the reflex on the hemiplegic side is exaggerated,

whereas the opposite one is normal. Or, again, it may occur in a beginning toxic neuritis or in a unilateral neuritis. It is, however, most commonly seen in cases of general paresis. So frequently does the condition occur in this disease that the student should never dismiss a case of unequal or irregular knee jerks without first eliminating a diagnosis of general paresis. This inequality of the knee jerks occurs with almost as much frequency as inequality and irregularity of the pupils, and is certainly as important.

There is no question but that in a few isolated cases the knee jerks are congenitally absent. I personally have known of two cases in which no knee jerk ever showed itself.

The **ankle reflex,** or **Achilles jerk,** is almost invariably present. E. Bramwell has advanced the theory that in persons over fifty years of age this reflex is often absent. Other observers, however, have not confirmed his view. To obtain the Achilles jerk have the patient kneel on a chair, with legs well separated. A slight tap just above the heel in a normal individual elicits an extension of the foot.

The segments of the spinal cord which control the Achilles jerk are the first and second sacral.

The main importance of the Achilles jerk lies in the fact that it of all reflexes is apt to be the earliest to disappear. Usually it is absent in those cases in which there is loss of the knee jerk, and sometimes several months in advance of the latter. This is especially so in tabes, alcoholic cases, and in general paresis. Thus, it will be seen that it is of value in foretelling rather than in actually diagnosing conditions.

THE ACHILLES JERK MAY BE ABSENT IN

1. Early tabes.
2. Peripheral neuritis.
3. Syphilis of the nervous system.
4. Sciatica.
5. Lesions of the lumbar and sacral regions.
6. Lesions of the cauda equina.
7. Infantile paralysis.
8. Diphtheria.
9. Diabetes.

Clonus is derived from the Greek word, κλόνος, meaning commotion. It is an involuntary, reflex, regular contraction of the muscles when suddenly put upon the stretch. It is a to-and-fro vibratory movement, and is never present in normal conditions. It is named, according to the part stimulated, ankle, knee, or foot clonus.

Functional conditions present a movement akin to it which is called spurious clonus. Of all the varieties, the most common is the ankle clonus. This phenomenon is due, as Weir Mitchell pointed out, to a contraction of the soleus muscle.

To obtain the **ankle clonus** have the patient either lie in bed or sit in a chair, while the examiner supports the flexed leg in a position of slight external rotation; with one hand under the calf, the other should grasp the distal part of the foot, and with the leg in a complete state of passiveness the foot should be suddenly flexed directly in a line with the leg. If under these conditions there should result a constant and regular series of to-and-fro movements, which continue as long as pressure on the foot is kept up, a true clonus is present.

To obtain **patellar clonus** have the patient relax

the extended leg when lying down, and then bring sudden and continued pressure on the patella downward.

The spurious ankle clonus met with in hysteria is not so regular, nor can it be maintained so long. The character of the contractions is also less pronounced; they lack an absolute simulation.

The cord segments which control clonus are the first three sacral.

The significance of the ankle clonus lies in the fact that its presence indicates a lesion of the pyramidal tract. It means that the cerebral inhibition has been withdrawn, and it is, therefore, one of the leading signs of paralysis of the upper motor neuron type.

DISEASES IN WHICH ANKLE CLONUS OCCURS

1. Spastic paralysis.
2. Hemiplegia.
3. Multiple sclerosis.
4. Brain tumor.
5. Cord tumor.

The **absence of a deep reflex** conveys to the examiner the information that in that particular reflex there has been an interruption; in short, a lost reflex means a break in the reflex arc. If the break is in the afferent fiber, there is, in addition to the loss of the reflex, a loss of sensation; if in the efferent fiber or in the anterior horns, there are muscular paralysis and atrophy. The site of the lesions, therefore, in the loss of the deep reflexes may be:

1. Peripheral sensory nerves.
2. Reflex centers of the cord.
3. Anterior horns of the cord.

4. Peripheral motor nerves.

The examination of the deep reflexes conveys to the examiner the information that either there has been (1) loss of the cerebral inhibition, that is to say, conveyance of the impulse from the brain to the cord has been interfered with, or (2) overstimulation of the spinal cord.

The conditions in which an exaggeration of the deep reflexes occur are:

1. Lesions of the pyramidal tract.
2. Poisons, as alcohol and arsenic.

The actual diseases are:

1. Cerebral thrombus and hemorrhage.
2. Lateral sclerosis.
3. Ataxic paraplegia.
4. General paresis.
5. Myelitis.

Oppenheim says that diseases of the pyramidal tracts are the most common cause of exaggerated reflexes.

Diseases in which the Knee Jerk may be Retained and the Achilles Reflex Lost

1. Amyotrophic lateral sclerosis.
2. Paralysis of the posterior tibial nerve.
3. Sciatica.

Finally, the student should bear in mind the two following rules:

1. Exaggeration of the deep reflexes means a lesion of the corticospinal or upper motor neuron tract.

2. Loss of the deep reflexes means a lesion of the spinomuscular or lower motor neuron tract.

It is almost a rule that in disease the superficial and deep reflexes are rarely of the same character, that is, when the deep reflex is exaggerated the skin reflex is lost.

CHAPTER XII

VERTIGO

Vertigo is a frequent symptom in diseases of the nervous system. Dizziness is a mild form of vertigo. Cerebellar vertigo is the name applied to a particular form of vertigo—one in which the unsteadiness is unilateral.

Vertigo occurs in both the organic and the functional diseases. It should be divided into two large classes—subjective and objective.

In the subjective class the patient gives no outward manifestations of being giddy. He only complains of a sensation of spinning around. In neuroses and in diseases of the brain it assumes a place of considerable importance.

In the objective variety he is seen to stagger and has a sensation of external objects moving. This variety is best illustrated in alcoholism.

To examine properly a patient suffering from vertigo it is necessary to make an examination of the ears, the eyes, the blood-vessels, and the general nervous system. Vertigo is exceedingly difficult to classify, but for practical purposes it is best to adopt a classification based upon an etiological basis. This divides the symptom into eight different classes:

1. The vascular type—which is dependent upon cardiac, blood, and circulatory causes.

2. The auditory vertigo—which is due to disturbances of the auditory or eighth nerve.

3. The ocular vertigo—which is due to disturbances in the muscles of the eye.

4. Vertigo due to reflex irritation—as from the stomach, liver, intestine, and other viscera.

5. Vertigo due to diseases of the brain.

6. Vertigo due to spinal cord disease.

7. Vertigo due to toxic conditions.

8. The vertigo of purely neurotic origin.

1. The **vascular type of vertigo** may be due to weakness on the part of the heart as a main pump; to a weakness in the blood itself, either in the form of an anemia or a toxemia, that is to say, to some interference with the fluid which the pump is forcing, or, finally, to some disturbances in the arteries or tubes through which the pump is forcing the fluid. In this latter instance the vertigo may be a result of a thickening or tortuous condition of the arteries. A list of the diseases in which the vascular type of vertigo may be a symptom is as follows:

(*a*) The different varieties of cardiac affections—*i. e.*, pericarditis, endocarditis, myocarditis, and valvular disease.

(*b*) Anemias.

(*c*) Toxemias due to drugs, especially alcohol, ether, chloroform and nicotin, and to gouty and diabetic products.

(*d*) Arteriosclerosis.

(*e*) The apoplexies.

2. The **auditory type of vertigo** occurs in a large number of cases. It is caused by an injury to the auditory nerve which results in disturbance of the space sense, and by many mild forms of ear disease, such as anemia or hyperemia. Gowers considers a large proportion of verti-

goes to be auditory in origin. A list of the diseases in which the auditory form of vertigo occurs is as follows:

(*a*) Ménière's disease.

(*b*) Congested conditions of the ear.

(*c*) Anemic conditions of the ear.

(*d*) Mechanical conditions of the ear.

3. The **ocular types of vertigo** are those due to some interference either in the muscles of the eye or in the refraction. They are few in number. A list of conditions in which they occur is as follows:

(*a*) Astigmatism.

(*b*) Muscular asthenopia.

(*c*) Errors in refraction.

4. The **reflex type of vertigo** comprises a very large number of cases. This type is due to some disturbances in any one of the different viscera, such as the stomach, liver, or intestines. In this type the vertigo is more apt to be slight and to take the form of dizziness rather than that of a real vertigo.

5. The **vertigo due to disease of the brain** may vary from slight dizziness to marked unsteadiness, or even to the typical reeling or cerebellar vertigo. Vertigo as a symptom in brain tumors occurs in from one-third to one-half of the cases. Here it is probably caused by an interference with the auditory or ocular nerves, or by a disturbance of the center of equilibrium in the cerebellum. A list of brain diseases in which vertigo occurs is as follows:

(*a*) Tumors and abscesses of the cerebrum and cerebellum.

(*b*) Multiple sclerosis.

(*c*) Cerebral arteriosclerosis—often an expression of

general or localized arteriosclerosis and common in old people.

(*d*) Hemorrhage or thrombosis.

(*e*) Changes in blood-pressure or general brain-pressure. Vertigo is largely if not wholly due to an interference with the centers of equilibrium.

Vertigo is a common symptom of general paresis, and as such requires comment. The attacks are usually either apoplectic or epileptic in character. They may simulate the characteristics of petit mal, that is to say, be merely attacks of dizziness or lapse of memory.

In neurological conditions the most important type of vertigo is cerebellar vertigo. It occurs as a result of lesions in the cerebellum. The patient presents a peculiar condition of reeling or falling in a semicircle, and always in the same direction, that is to say, if a patient starts to walk across the room he will invariably end on the opposite side of the room several feet to the left or to the right, as the case may be, giving very much the same result as a boat would when carried by the tide. A patient with cerebellar vertigo rarely falls, rather does he constantly threaten to fall. Cerebellar vertigo cannot be mistaken for any other type of vertigo, as it is most intense, unilateral, and never ends in an absolute loss of balance. It is generally accompanied by nystagmus. The conditions which may cause cerebellar vertigo are:

(*a*) Localized lesions of the cerebellum, resulting from tumors, hemorrhages, patches of sclerotic tissue, etc.

(*b*) Congenital defective cerebellar development.

(*c*) Hereditary cerebellar ataxia.

(*d*) Gerlier's disease.

The importance of vertigo is not so great in diagnosis as in prognosis.

In epilepsy vertigo is a prominent symptom. It may vary from complete loss of consciousness in grand mal to slight dizziness in petit mal.

Vertigo is a very frequent symptom in syphilitic conditions of the nervous system, such as tabes, paresis, and cerebrospinal syphilis.

6. **Vertigo due to spinal cord disease** is unusual and a symptom of no importance.

7. **Vertigo due to toxic conditions** is most commonly seen in alcoholism. It may also result from ether, chloroform, nicotin, and other drugs; it may arise from gouty, intestinal, or diabetic products. The drugs poison the nerve cells in the cerebellum. In these instances the vertigo is intense and partakes of the nature of a bucking or intense type.

8. **The Vertigo of Purely Neurotic Origin.**—This type of vertigo is psychic in origin; it occurs in all of the functional conditions, as neurasthenia, hysteria, hypochondria, and the traumatic neuroses. It is rarely attended by staggering, but most often by dizziness. It is akin to the phobias or morbid fears of which so many patients complain. The best illustration of this type is the dizziness felt when looking down from heights.

The vertigo of purely neurotic origin assumes many different types varying from a slight transient sense of dizziness to a marked vertigo. It is most apt to occur in the morning, after prolonged work, a great strain, or emotional excitement of any kind; it may follow absence of fresh air or occur without apparent reason. A list of conditions in which this type of vertigo occurs is:

(*a*) Hysteria.

(*b*) Various degrees of emotional and nervous stress.

(*c*) Psychasthenia.

(*d*) Nervous headache, migraine.

(*e*) Excessive nausea or vomiting.

List of conditions in which vertigo is a symptom:

1. Tumors, abscesses, and other lesions which increase intracranial pressure.
2. Multiple sclerosis.
3. Arteriosclerosis and endarteritis.
4. Meningitis.
5. General paresis.
6. Tabes.
7. Myasthenia gravis.
8. Ménières disease.
9. Cranial nerve paralysis.
10. Lowered blood-pressure.
11. Toxic conditions.
12. Ataxia.
13. Caisson disease.
14. Migraine.
15. Increased blood-pressure.
16. Psychical vertigo.
17. Reflex vertigoes.
18. Laryngeal vertigoes.
19. Intracranial circulatory anemia.
20. Overdoses of quinin.
21. Vertigo of antrum of Highmore, ethmoidal or sphenoidal diseases of sinus.
22. Orbital congestion.
23. Neurasthenia.
24. Hysteria.
25. Cerebral endarteritis.

CHAPTER XIII

THE EYE

Examination of the eye affords threefold information: First, it gives an insight into the condition of certain of the cranial nerves; second, information as regards certain reflexes, and, third, information as to the ingestion effects of certain toxins and drugs. Here, as well as in gaits, inspection will greatly reward the student and afford wide information. It will be very advantageous to acquire the habit of observing a patient's pupils. This can be done when the patient first presents himself. By standing directly in front of the subject and then moving a little to one side, it is possible to throw upon the pupil first darkness and then light. In this way it is possible to diagnose Argyll Robertson pupils, irregular pupils, unequal and very much dilated pupils. A practical point worth remembering, though not one of universal application, is that a markedly dilated pupil is frequently symptomatic of functional disease, whereas a markedly contracted pupil is symptomatic of organic disease. Another point to remember is that a large number of organic affections, both of pupils and of muscles that control the eye, are the result of syphilis.

It is best to consider the relation of the eye to nervous diseases under three headings:

A. The physical signs of the pupil.

B. The physical signs aside from the pupil.

C. The eye reflexes.

A. The physical signs of the pupil:

1. Miosis.
2. Mydriasis.
3. Inequality or anisocoria.
4. Irregularity.
5. Immobility.
6. Hippus.

B. The physical signs aside from the pupil:

1. Ophthalmoplegia.
2. Optic neuritis.
3. Choked disc.
4. Optic nerve atrophy.
5. Ptosis.
6. Diplopia.
7. Nystagmus.
8. Conjugate motion of the eyes.
9. Hemianopsia.
10. Achromatopsia.
11. Dyschromatopsia.
12. Scotoma.
13. Hemeralopia.
14. Interstitial keratitis.
15. Graefe's sign.
16. Stellwag's sign.

C. The eye reflexes:

1. The Argyll Robertson pupil.
2. The consensual reflex.
3. Wernicke's hemiopic pupillary reaction.
4. Ciliospinal reflex.
5. Associated movement of iris in accommodation.

Physical Signs of the Pupil

Miosis is contraction of the pupil. It is one of the two forms of pathologic alteration in the diameter of the pupil. Another name for it and one by which it is often known is that of "pinpoint" pupil.

The lesion in miosis is situated in the sphincter inhibitory center, or in the fibers coming from it.

There are two types: spastic miosis and paralytic miosis.

In the spastic form there is contraction of the sphincter iridis by stimulation; in the paralytic form there is paralysis of the dilator pupillæ.

Miosis is a physical sign in these conditions:

Spastic or Irritative Miosis	Paralytic Miosis
Cerebral tumor (early stages).	Tabes.
Meningitis.	General paresis.
Cerebral abscess.	Cerebral syphilis.
Apoplexy.	Certain drugs.

Mydriasis is dilatation of the pupil. It, with miosis, comprises the only forms of pathologic alteration in the diameter of the pupil.

Mydriasis may result from several causes, such as:

1. Impulses to the ciliospinal center in the cord, coming either from the brain or from any region of the body.
2. A rundown physical condition.
3. Any irritation of the medulla exciting the respiratory centers.
4. Emotional causes.
5. Stimulation of the sympathetic fibers in the neck.

Mydriasis, like miosis, should be regarded as irritative or paralytic:

Irritative:

An irritative lesion in the upper part of the spinal cord.

An irritative lesion of the sympathetic dilating fiber in the neck.

Paralytic:

Intracranial pressure.

Lesions of part of third-nerve nucleus.

Anisocoria is a synonym for inequality. It is a condition in which the pupils are of different sizes, without regard to regularity of outline or mobility. It may occur in diseases of the stomach, lungs, kidneys, liver, and arteries, such as inflammation of the pleura, apical tuberculosis, pneumonia, and aneurysm. It is especially common in neurologic conditions.

Conditions in which Anisocoria may Occur

Congenital affections of the sympathetic.

Different refractions of the two eyes.

Unequal illumination of the two eyes.

A lesion in only one centripetal light-reflex tract.

A lesion in only one centrifugal light-reflex tract.

Aneurysm.

Inequality of the pupils may occur in several organic and toxic nervous conditions; it is especially common in—

Locomotor ataxia.

General paresis.

Cerebral syphilis.

Alcoholism.

The unequal pupil should, therefore, be regarded as strongly indicative of the presence of syphilis.

Irregularity is a condition in which the contour of the

pupil is not uniform; it is often confused with inequality, but is entirely different. Pupils may be both irregular and unequal. Unequal pupils appear: Irregular pupils: Irregularity is one of the physical signs of a syphilitic pupil.

Immobility is a condition in which the pupil fails to respond to the light reflex and remains immobile. It is a loss of the light reflex, and is akin to the Argyll Robertson pupil. This condition is caused by a break in the reflex arc of the pupil. A pupil which responds to light slower than the normal, and yet is not absolutely immobile, is called "sluggish."

These three conditions of inequality, irregularity, and immobility are particularly characteristic of a pupil resulting from syphilis. The combination of the three, then, is especially to be found in general paresis, tabes, and cerebral syphilis. As all three begin with I, and all relate to the symptoms of the eye, to remember them is easy·

Irregularity.
Inequality.
Immobility.

Hippus is a condition in which there is an alternate contraction and dilatation of the pupil. As one observes the pupil he sees it change constantly from contraction to dilatation and vice versâ. The condition is especially seen in mania, anemia, and some of the functional conditions.

Physical Signs Aside from the Pupil

Ophthalmoplegia is a syndrome; it is the name given to a paralysis of certain ocular muscles. When the external muscles of the eyeball, *i. e.*, those supplied by the

Fig. 49.—Double ophthalmoplegia.

Fig. 50.—Double ophthalmoplegia.

third, fourth, and sixth nerves, are paralyzed, an ophthalmoplegia *externa* occurs. When the muscles of the iris and pupil are paralyzed, an ophthalmoplegia *interna* occurs.

Ophthalmoplegia externa means that the action of the eyeball is involved; ophthalmoplegia interna means that the action of the pupil is involved, that is, the iris and ciliary muscle. Either type may be acute or chronic, single or double; there may even be a mixed type. The latter is a very rare condition.

Causes of Ophthalmoplegia Externa

1. Chronic alcoholism.
2. Hemorrhage.
3. Embolus and thrombosis.
4. Tumors.
5. Multiple sclerosis.
6. Tabes, general paresis, and syphilitic exudations.
7. Toxemia.
8. Poisoning (lead and nicotin).
9. Exophthalmic goiter.
10. Diseases of the medulla.
11. Traumatism.
12. Diabetes.
13. Tubercular meningitis.

Causes of Ophthalmoplegia Interna

1. Infection:
    - (*a*) Syphilis (most frequent cause).
    - (*b*) Influenza.
    - (*c*) Acute rheumatism (rare).
    - (*d*) Scarlet fever (rare).
    - (*e*) Measles (rare).
    - (*f*) Erysipelas (rare).

(*g*) Pneumonia (rare).
(*h*) Mumps (rare).
(*i*) Typhoid (rare).
(*j*) Zoster.
(*k*) Postdiphtheritic.
(*l*) Tuberculosis.

2. Exostoses and syphilitic periostitis.
3. Thrombosis of sinus cavernosus.
4. Toxic origin:
   (*a*) Alcoholism (chronic).
   (*b*) Diabetes.
   (*c*) Acute nephritis.
   (*d*) Saturnine poisoning.
   (*e*) Fish, meat, and oyster poisoning.
   (*f*) Aconite, atropin poisoning.
   (*g*) Mushroom poisoning.

Optic Neuritis.—The condition known as optic neuritis consists of a swelling of the optic disc, which is of grayish color, striated, with a blurring of the margin; a swollen, tortuous condition of the veins, with a constriction of the arteries. It begins as a papillitis, and, when it has progressed a few degrees, becomes a choked disc. Dana defines the choked disc as a papillitis with much serous infiltration. Remember, optic neuritis and choked disc differ in degree only. Therefore, cases will occur in which what to one man is an optic neuritis to another is a choked disc.

In optic neuritis there are less swelling and less projection of the disc. There is, however, more exudation, and this extends more beyond the disc into the retina.

In choked disc the swelling is limited to the disc; the predominant characteristics are the engorgement and

the edema. Of the two, inflammation is more pronounced in neuritis.

In either form sight may or may not be impaired. The more advanced the neuritis, the greater is the likelihood of a defect in the vision. The most frequent and most

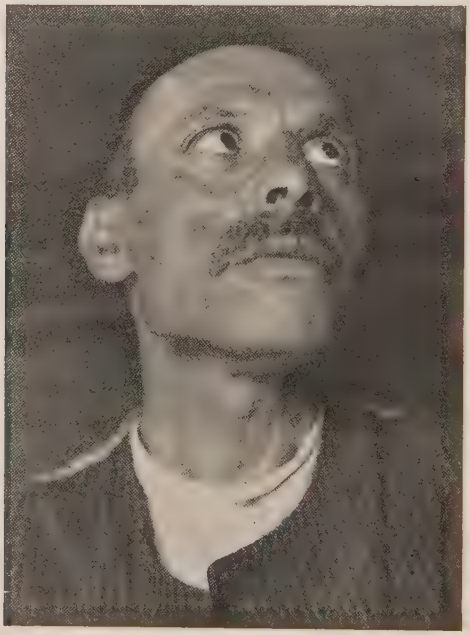


Fig. 51.—Sixth nerve palsy.

important cause of these two conditions—conditions which are really the same but different in degree—is tumor of the brain. Sir William Gowers says that in four out of five cases of brain tumor optic neuritis is present. Wyllie says in cerebellar tumors the proportion is fifteen in sixteen.

Optic neuritis may affect one or both eyes; it may develop early or late; it may advance rapidly or slowly. The type which accompanies the cerebral tumor and tumors of the cerebellum is the same.

Optic neuritis, or, to use the better term, papillitis, occurs in the following conditions:

1. Heredity.
2. Orbital and peri-orbital affections.
3. Brain tumors and abscess.
4. Hydrocephalus.
5. Meningitis.
6. Infectious fevers.
7. Nephritis.
8. Toxic neuritis.
9. Alcohol.
10. Lead poisoning.
11. Severe hemorrhage.
12. Diseases of menstruation, pregnancy, and lactation.
13. Idiopathic.

The most frequent cause is tumor of the brain. Next to this comes meningitis.

The form of papillitis most common in brain tumors begins as an optic neuritis, later becoming a choked disc.

These conditions of papillitis, optic neuritis, and choked disc can be observed and diagnosed only by the ophthalmoscope. The skilled user of this instrument can with a little practice measure the amount of swelling of the disc. The diameter of the disc may be even two or three times its normal size.

Choked Disc.—Von Graefe first gave us the term "choked disc." Its origin has never been made clear.

Two theories have been suggested to explain it: one is pressure, edema, and inflammation; the other is toxemia.

Fig. 52.—Hernia cerebri following decompression.

The final stage that choked disc attains is **optic nerve atrophy.** Therefore, a patient suffering from a choked disc or optic neuritis requires constant observation; if

the condition advances, surgical interference may be necessary to relieve pressure on the optic nerve and thus save the sight.

Optic Nerve Atrophy.—In this condition there is degeneration of the optic nerve, with resulting atrophy and loss of vision.

There are two types:

1. Primary optic nerve atrophy, or simple.
2. Secondary optic nerve atrophy, or inflammatory.

1. *Primary optic nerve atrophy* is sometimes called simple atrophy. In this form the disc is white, with sharply defined edges and a saucer-shaped excavation. The size is somewhat diminished. The lamina cribrosa is often seen very plainly; the minute vessels have disappeared; the arteries of the retina are diminished in caliber.

Simple atrophy is due to:

1. Syphilis, general paresis, and tabes.
2. Tumors.
3. Multiple sclerosis.
4. Malaria.
5. Diabetes.
6. Certain poisons, *i. e.*, wood alcohol, lead.
7. Myxedema.
8. Hemorrhage.
9. Idiopathic.

2. *Secondary atrophy* is also called postneuritic atrophy. In this form the disc is white, but the edges are irregular. There is no saucer-like depression, and the disc is covered with new connective tissue from the previous neuritis, which hides the lamina cribrosa.

Secondary atrophy is due to:

1. Papillitis.
2. Retrobulbar neuritis.
3. Retinitis.

Fig. 53.—Ptosis.

4. Embolism of the central artery.
5. Glaucoma.
6. Pigmentary degeneration of the retina.
7. Fracture of the orbital canal, causing wounds to nerve.

Ptosis.—The term ptosis means drooping of the eyelid. It results from paralysis of the third nerve. In addition to drooping of the lid there occurs double vision, divergent strabismus, dilatation of the pupil, loss of the light reflex, and loss of the power of accommodation. All muscles supplied by the third nerve usually are not attacked simultaneously. It may occur in all degrees and may be single or double. Unilateral ptosis is most frequently syphilitic in origin. The causes are:

1. Syphilis.
2. Congenital.
3. Traumatism.
4. Non-development of the nucleus of the third nerve.
5. Postdiphtheritic.
6. Epidemic cerebrospinal meningitis.
7. Polio-encephalitis hæmorrhagica superior.
8. Chronic progressive ophthalmoplegia.
9. Intoxications.
10. Multiple sclerosis.
11. Tabes.
12. General paresis.
13. Functional disturbances.
14. Syringomyelia.
15. Brain tumor.
16. Neurasthenia.
17. Myasthenia gravis.

Diplopia means double vision. It is due to the fact that the images of the object fall on non-corresponding fields of the retina.

Diplopia occurs in the following conditions:

1. Tabes.
2. General paresis.

3. Cerebrospinal syphilis.
4. Alcoholism.
5. Brain tumor.
6. Myxedema.
7. Hysteria.

The cause of diplopia is a paralysis of certain eye-muscles.

Diplopia may be of two kinds: *homonymous*, when the false image is on the same side as the sound eye; *heteronymous*, when the false image is on the opposite side.

Diplopia is a symptom of:

1. Oculomotor, or third nerve, palsy.
2. Abducens, or sixth nerve, palsy.
3. Pathetic, or fourth nerve, palsy.

Nystagmus is an oscillation of the eyeball. The best illustration is the eye of a person observing passing objects from a rapidly moving train.

The oscillations are rapid and the extent of their excursions is short. The direction of these oscillations may be any one of several. Therefore, nystagmus may be lateral, vertical, mixed, or rotatory. The condition is nearly always bilateral.

Nystagmus may be physiologic or pathologic, and occurs in the following conditions:

1. Physiologic nystagmus.
   - (*a*) Person riding in a train.
   - (*b*) Person suddenly whirled around.
   - (*c*) Galvanism.
   - (*d*) Irritations of the inner ear by alternate hot and cold irrigations.
2. Pathologic nystagmus.
   - (*a*) Congenital amblyopia; error of refraction.

(*b*) Sudden anemia or disorders of the cerebral circulation.
(*c*) Certain conditions of intoxication.
(*d*) Certain brain lesions: affections of the cerebellum; affections of the cerebellar peduncle, the nuclei of the oblongata, the posterior longitudinal bundle, the vestibular nerve.
(*e*) Certain occupations: miners.
(*f*) Disseminated sclerosis.
(*g*) Meningitis.
(*h*) Hereditary ataxia.
(*i*) Reflex symptom.
(*j*) Reflex symptom from remote irritation.

Diseases in which Nystagmus Occurs

1. Multiple sclerosis.
2. Friedreich's ataxia
3. Alcoholism.
4. Syringomyelia.
5. Brain tumor.
6. Heredity.
7. Meningitis.
8. Cerebral hemorrhage and softening.
9. Cerebral and lenticular opacities (choroiditis and retinitis).
10. Albinism.

Nystagmus is elicited by having the patient's eye follow the examiner's finger as he holds it well out to one side. Pathologic nystagmus is not present unless apparent during the movement of the eye, as the oscillations which appear after the eye is at rest in the

extreme position may be physiologic and the result of fatigue.

The occurrence of nystagmus in diseases of the spinal cord is without doubt an indication of the involvement of the cerebrospinal nervous system or the cerebellum or the parts adjoining it.

Conjugate Motion of the Eye.—The two eyes move together. They may converge to observe a near object; they may diverge to observe a distant one; they may turn up or down. In any event in the normal individual they move together. The mechanism necessary to bring about this condition is complex.

Some clinicians maintain that:

Destructive lesions of the brain cause the patient to look *toward* the lesion.

Irritative lesions of the brain cause the patient to *look away* from the lesion.

Hemianopsia means absence of half the field of vision of each eye. There are two varieties: (*a*) homonymous, when it affects the corresponding halves of the eyes; (*b*) heteronymous, when it affects the opposing halves of the eyes. Heteronymous is of two kinds: bitemporal and binasal. Thus, a blindness of the two outer halves of the eyes would be bitemporal hemianopsia, whereas a blindness of the outer half of the right and the inner half of the left retina would be a left homonymous hemianopsia. These various forms depend upon the location of the lesion.

The causes of bitemporal heteronymous hemianopsia are tumors at the base, tumors of the hypophysis, and syphilis at the base of the brain.

The three rules to remember in regard to hemianopsia are as follows:

1. If the blindness is restricted to one eye or affects both eyes, but is not symmetric, then the lesion must be in the optic nerve itself.

2. In homonymous hemianopsia the lesion is above the chiasm.

3. In bitemporal hemianopsia the lesion is in the chiasm itself, located anteroposteriorly. This is seen in acromegaly.

Hemianopsia is almost invariably a symptom of an organic disease. It may, however, rarely be found in certain functional conditions and in migraine and gout.

The organic conditions in which hemianopsia occurs are as follows:

1. Hemorrhage.
2. Inflammation.
3. Tumors.
4. Softening.

The test for it is to have the patient look steadily and straight at the examiner; meanwhile the latter brings some small object from the extreme right and left into the field of vision. The patient, if suffering from hemianopsia, will not be aware of its presence until it has traversed one-half of the visual field. A second test is that the eyelid will not wink at the close proximity of the finger to the blind half of the eye.

Achromatopsia means loss of color vision.

Dyschromatopsia means imperfect color sense.

Scotoma is a blind spot. It may be on the periphery of the visual field or in the center; in accordance with its location, is known as peripheral or central sco-

toma. It should be tested for by moving a small piece of paper while the patient looks straight ahead at a fixed point, one eye being closed until the test paper reaches a point in the field of vision at which it appears dull, colorless, or is invisible. Pathologically patients notice the defects in the field of vision.

There may be central scotomata only in respect to certain colors—a common symptom in tobacco poisoning.

Diseases in which a Central Scotoma Occurs

1. Organic diseases of the optic nerve { Atrophy. / Hemorrhage.
2. Organic diseases of the retina.
3. Central opacities in lens or cornea.
4. Multiple neuritis.
5. Migraine.
6. Partial destruction of peripheral optic tract.
7. Tobacco and alcohol poisoning.
8. Multiple sclerosis.

Hemeralopia is day-blindness. This is a condition in in which the patient is able to see better in a dim light. It is a symptom which occurs along with amblyopia and is common in nicotin poisoning. Stewart says that "it is probably due to the fact that the bright light rapidly fatigues the retina, and by producing pupillary contraction causes the peripheral part of the retina to be less in use than the central, whereas in the dim light the pupil dilates and the unaffected peripheral portion of the retina comes into play."

Von Graefe's Sign.—The upper eyelid does not smoothly accompany the eyeball when the patient's eye follows the finger from above downward: it lags.

Stellwag's Sign.—A widening of the palpebral fissure, together with a decrease in the frequency of winking, is known as Stellwag's sign.

THE EYE REFLEXES

In the **Argyll Robertson pupil** the reflex to light is lost, though the reflex to accommodation is retained. The condition is usually bilateral; it may be unilateral. It is caused by a break in the reflex arc. To impress this remember the two last letters of the word *Argyll* (*l. l.*), meaning loss of light reflex, and the first two letters (*a. r.*), meaning accommodation retained.

The way to test for the symptom is to have the patient look at a fixed object, shade the eye to be examined, and then suddenly admit light to the pupil. In health the pupil should at once contract. A good light should always be used; at times an electric light is best. Most of the internes in the hospitals carry small pocket electric light lamps. With this method a good light can be quickly admitted to or removed from the pupil. Another method of examining the pupil is to throw a light into the eye by means of an ophthalmoscopic mirror; still another, by means of a lens. Judgment on a pupil should never be pronounced until it has been tested by a good light.

It should always be borne in mind that no hard and fast rule can be laid down. There is, in the case of the reflexes of the pupil, as in every other instance, every degree of gradation from the normal to the pathologic. Previous to the pupil becoming rigid, there is a stage in which the reaction is slow or, as it is technically called, "sluggish." The sluggish type of pupil is an incipient

Argyll Robertson pupil, and one which becomes so after a time. The sluggish pupil may precede the Argyll Robertson by months or even years.

The Argyll Robertson pupil is a symptom of:

1. Tabes.
2. General paralysis.
3. Progressive muscular atrophy (Charcot-Marie-Tooth type).
4. Disseminated sclerosis.
5. Cerebral softening or atrophy.
6. Cerebral syphilis.
7. Hydrocephalus.
8. Tumors of third ventricle.
9. Congenital syphilis.
10. Alcoholism.

Sluggish pupils occur in—

1. Filix-mas poisoning.
2. Chloroform narcosis.
3. Acute alcoholism.
4. Acute morphinism.

It is, however, most common as a symptom of:

1. Tabes.
2. General paresis.
3. Cerebral syphilis.

The Argyll Robertson pupil is a most valuable sign in the diagnosis of tabes, in which it occurs in 90 to 95 per cent. of the cases and in general paresis.

Another name for it is reflex iridoplegia.

The Consensual Reflex.—The consensual reaction is the reaction to light, which occurs in one pupil as the result of the admission of light to the other. To test for this reflex, shade and then illuminate the eye to be ex-

amined while at the same time we note the light reaction in the other. Immobility in either eye proves a deficient light conduction, and therefore an absence of the consensual reflex. This is directly opposed to the direct reflex, which simply means that the pupil examined is susceptible to light and to darkness.

Wernicke's Hemiopic Pupillary Reaction.—A ray of light thrown on the blinded halves of the retinas (in homonymous lateral hemianopsia) causes no contraction of the pupil if the lesion is peripheral to the point at which the reflex fibers to the Edinger-Westphal pupillary nucleus are given off. Illumination of the sensitive halves of the retinas does, on the contrary, cause contraction of the pupil if the lesion is central to the point at which the above reflex fibers are given off.

The **ciliospinal reflex** consists of the dilatation of the pupil caused by stimulation of the skin of the neck on the same side.

CHAPTER XIV

DISTURBANCES OF SPEECH

Just as certain neurologic conditions have gaits and tremors that are characteristic and diagnostic, so others exhibit peculiar forms of speech and expression in which not only expression, but also tone of voice, is altered.

To examine the patient's speech it is necessary first to have him speak, next to have him read aloud, and lastly to have him repeat some test phrase. It is best to examine his speaking ability while he is telling the history of the case, so that his attention will be distracted from what the examiner is trying to discover. In reading aloud the patient should be given some simple matter, preferably the daily paper. In repeating the catch phrases he should be given ample opportunity to hear and comprehend the significance of the words, and then be asked to repeat after the examiner. There are many phrases which are used. It is best to have two or three and always to make use of the same ones. In this way the examiner gets accustomed to the various pronunciations of set phrases: "Third riding artillery brigade," "Methodist Episcopal," and "Hippopotamus hurrying home." These phrases should always be repeated three times in succession.

The normal speech is free from any idiosyncrasy. There is no blurring or elision of syllables. It is noteworthy neither for slowness nor for rapidity. The words

are expressed in a clear, straightforward manner. The tongue and lips are free from any tremor.

In testing speech it is important to observe the manner of expression, the speed with which the patient talks, the clearness with which he pronounces the different words, and the absence of any elision or slurring. Note whether the pitch of the voice is high or low.

I. Dysarthria, or disturbances in articulation.

A. Varieties:

1. Disturbances in phonation: vowels and consonants indistinct.
2. Disturbances due to paralysis of lips, tongue, or palate muscles; nasal voice and as if patient had pudding in mouth.
3. Bulbar speech.
4. Anarthria.
5. Bradylalia and scanning speech.
6. Stuttering.
7. Lisping: negligent, organic, neurotic.
8. Stumbling speech.
9. Slurring speech.

B. Causes:

1. Due to toxic action of alcohol; early stage of ether anesthesia.
2. Due to lack of development and infantile disease:
   Idiocy and cretinism.
   Diplegia.
   Hereditary ataxia.
3. Due to local disturbances:
   Cleft-palate.
   Hare-lip.
   Absence of teeth.
   Perforated nasal septum.
   Stomatitis.

4. Due to disease:
   - Multiple sclerosis.
   - General paresis.
   - Brain tumor.
   - Hemiplegia.
   - Myasthenia gravis.
   - Chorea.
   - Epilepsy.
   - Bulbar palsy.
   - Paralysis agitans.
   - Insanity (senile and manic).
   - Meningitis.
   - Progressive lenticular degeneration (Wilson's disease).
   - Myxedema.
   - Friedreich's disease.
   - Diplegia.

II. Aphonia.
   - A. Due to hysteria.
   - B. Due to paralysis of vocal cords.
   - C. Due to paralysis of muscles of larynx.
   - D. Due to tumor of larynx.

III. Aphasia.
   - A. Motor.
   - B. Sensory.

Varieties of Dysarthria

There are many kinds of disturbances in articulation. A list of the principal ones stands at the head of this chapter. Here I shall say only a word or two in describing the most important.

The simplest form of all is that which results from an *indistinct manner of pronouncing* the various letters, both vowels and consonants.

The second type is the one resulting from a *paralysis*

of the several muscles concerned in the act of speaking. This type varies according to which muscles are involved: if the lip muscles alone are involved, the defect is evident in the labials and in the labiodentals. If the tongue only is involved the defect is evident in the linguals and in the appearance of the tongue itself, which is shriveled, tremulous, and protrudes with difficulty. If the palate alone is involved, it is evident in the palatals and in the nasal twang. There may exist any combination of these conditions.

Bulbar speech is the name given to that type of speech which accompanies lesions of the bulb, that is, lesions of the pons and medulla. This is fully described elsewhere.

Anarthria is the term applied to that form of speech which is absolutely unintelligible. It is neither aphasia nor aphonia. In anarthria the patient can emit sounds, but they convey no sense.

Bradylalia is slowness of speech. It is characteristic of some imbeciles, of the child learning to speak, and even of some normal people, who cerebrate with great slowness. *Scanning speech* is placed under this heading because of its slowness; however, its most marked characteristic is the great deliberation and clearness with which each syllable is enunciated. The sound conveyed to the listener is that of a person scanning a line of Virgil. This scanning speech is described later on. It occurs most commonly in multiple sclerosis; it may, however, accompany general paresis and some forms of aphasia.

Stuttering is a spasmodic form of speech disorder. The muscles of speech are affected with cramps or spasms.

Lisping, according to Scripture, may be of three kinds. The negligent type is due to defective perception and

execution of sounds; the organic type is due to abnormal condition of the speech organs; the neurotic type is due to functional disturbances and the temperament of the patient.

Slurring speech is synonymous with thickness of speech. It is especially evident following protracted and severe illness, such as typhoid.

Causes of Dysarthria

The speech of the alcoholic varies from simple garrulousness to complete paralysis. The release of motor impulses is greatly accelerated, with the result that the patient repeats those phrases which are most familiar and which he has long since learned by heart. He is apt to make use of compound words and rhymes.

In the lowest forms of *idiocy*, babbling, grunting, and echolalia are the characteristics, and also the use of words of the patient's invention. Lalling and stammering are of frequent occurrence, but are more likely to be met with as we ascend the scale. *The speech of the cretin* has neither characteristic difficulty nor unusual tone. It is slow, labored, and develops late. The vocabulary is limited.

The speech of diplegia is slow and stammering. If the condition appears in the very young, speech may never develop; if in older children, the result is usually a gradual loss of speech.

The speech of Friedreich's disease, or *hereditary ataxia*, is slow, ataxic, and scanning. It resembles neither the pure type of multiple sclerosis nor that of paresis; it is rather a combination of the two; some words come quickly, others slowly. The pitch of the voice is apt to

change suddenly and in the midst of a sentence. As the disease advances, a mental enfeeblement becomes evident, which serves further to modify the articulation. This speech is clumsy, and conveys to the listener the impression that the patient has a mouthful of something very hot.

The most common defects of speech due to **local disturbances** are those resulting from *hare-lip* and *cleft-palate*. In the former there is an interference with the proper pronunciation of the labials, while in the latter there is an interference of the palatals, accompanied by a nasal twang.

The speech of multiple sclerosis is peculiarly diagnostic. While the tone is monotonous, the enunciation of the words is clear and measured. It can best be designated by the word "scanning," as its tones recall the scanning of a line of Virgil. Scripture describes it as bringing out each syllable with a distinct effort, but without the characteristic anxiety of the stutterer. Oppenheim says that the patient speaks like a child spelling out letters. Another word that has been used in connection with it is "staccato."

The Speech of General Paresis.—The words which best describe the speech of the paretic are ataxic, stumbling, and alliterative. There is a marked tendency to slight words, and even syllables. The speech is slow, halting, and at times stops altogether. It can be compared to the patient's gait. It is irregular, stumbling, and uncertain. Accompanying the mechanical defect in speech is a loss of memory, together with a progressive enfeeblement of mind and a tremor of the muscles about the mouth. These factors complicate the picture and pre-

sent a form of speech which is both cortical and peripheral. This is the one form of speech which is most clearly illustrated by use of the test phrases.

In *brain tumors* the speech may or may not be affected, according to the location of the growth. If it is in the third left frontal convolution so as to affect the speech center, all varieties of disturbances will arise. At first there may be only a slight disinclination to speak; later on mild aphasia will develop, and eventually complete loss of speech. The prevailing hebetude, which is so diagnostic of these patients, becomes evident in the speech. The patient is slow to think, slow to talk, and speaks with difficulty. If the lesion is in other parts of the frontal convolution there may be no disturbance.

If the lesion is in the left temporal lobe, there will be word-deafness, amnesic aphasia, and paraphasia.

If the lesion is in the left inferior parietal lobe, there will be alexia and agraphia.

It must always be borne in mind that if the patient is left-handed a tumor situated in the right hemisphere will give all the symptoms of one in the left hemisphere of a right-handed man.

The speech of hemiplegia is often involved. The character of the defects differs according to the location of the lesion. If the paralysis is on the right side, the lesion on the left, the speech center is involved, and there may result one of the many forms of aphasia, apraxia, or alexia. If, on the other hand, the paralysis is on the left, the lesion on the right, there is no involvement of the speech center. The difficulty is one of articulation rather than of speech. In this instance the voice is thick and indistinct; the linguals are improperly pronounced, and,

from the physical interference which the paralyzed half of the tongue, lips, and cheek muscles causes, the enunciation is slurring, indistinct, and thick. It should al-

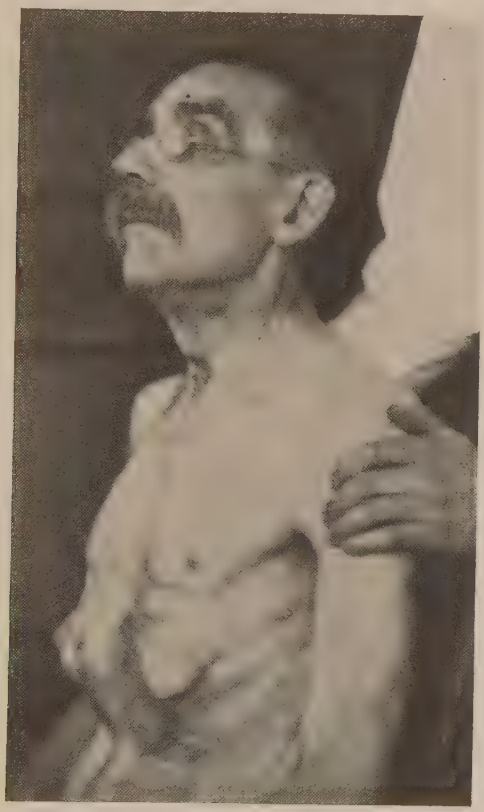


Fig. 54.—A case of tabes showing double ptosis and nodule deformities at the costochondro-articulations (Charcot ribs).

ways be borne in mind that this condition of affairs may be completely reversed, as in left-handed persons the speech center is on the right side of the brain.

The speech of myasthenia gravis is variable. It may remain normal. It may, as a result of the weakening of the muscles, slowly and gradually become more and more indistinct, until it is lost altogether; it may simulate that of bulbar palsy. The principal feature is a progressive tendency to grow fainter, feebler, and more indistinct, until following a period of rest it again becomes normal.

The speech of the choreas is jerky and irregular. The words are broken, and, being affected by the breathing movements, come forth in a violent and aggressive manner. They are hurled forth. This defect of speech is more evident in the chronic forms, but to a slight degree does exist in the acute types. In the advanced and aggravated cases the patient may be unable to speak at all.

The speech of epilepsy is neither characteristic nor distinctive, although in some cases monotonous. In many instances the enunciation is thick and indistinct; this is due to the fact that the patient is suffering more from overdoses of bromid than from epilepsy. As a postepileptic manifestation there often develop disturbances which are central in origin and accompany mental deterioration.

The speech of bulbar palsy is nasal. It becomes gradually indistinct, more labored, and finally unintelligible. The principal mechanical difficulty is inability on the part of the patient to pronounce the dentals and linguals. Added to this is an inability to express the words. The paralysis of the throat, tongue, and lips renders it exceedingly difficult for the patient to get the words out of his mouth. He is, therefore, forced to make extraordinary efforts, and so conveys the impression of one

making an effort beyond his powers, and raising his voice to an unnecessarily high pitch. The voice is usually nasal; it may be hoarse, and in the aggravated cases is explosive. Added to all these difficulties are a dribbling of saliva, a tremor of the lips, a nasal regurgitation of fluids, and a difficulty in swallowing and coughing.

The speech of paralysis agitans can best be compared with the gait of paralysis agitans. As the patient festinates in his walk, so does he festinate in his talk. He is slow to begin, the lips are opened, great deliberation is displayed, there is hesitancy, and at last, slowly and deliberately, the first few words make their appearance. Then he speaks faster and faster until finally the words seem to tumble out after each other, so rapid and explosive is the utterance. Another feature of this condition is the change of voice. It becomes shrill, high-pitched, and piping.

In *myxedema* the speech becomes slow, rough, and monotonous.

Besides this list there are also the different brain conditions which produce the aphasias. I have not included them in this category, as I regard them as being of sufficient importance to occupy a place of their own. Aphasia, alexia, apraxia, etc., have always been confusing to students, and ought, therefore, to be in a class by themselves.

CHAPTER XV

APHASIA

The word aphasia is derived from the Greek α, privative, and φημί, which means to speak. The technical meaning of the word, therefore, is absence of speaking. Webster gives an excellent explanation of the term when he defines it as "loss of the power of speech or of the appropriate use of words, the vocal organs remaining intact, and the intelligence being preserved." In other words, the aphasic is one who is affected with a lesion in one of his speech centers, and yet, at the same time, is perfectly conscious of his infirmity. These speech centers are situated in the cortex of the brain. The fibers that connect them are subcortical.

The subject of aphasia has always been exceedingly confusing and difficult to the student. This, to a great extent, has been the fault of the text-books and the teachers. There has never been any definite agreement among authors as to the proper and uniform division of the subject. The only way to understand the subject of aphasia is to look at it from the simplest point of view, to apply to it as few and as simple terms as possible, and to divide and subdivide it in a systematic, common-sense way. Looked at and studied under these conditions, it ceases to be the bugbear which students always picture it; there emerges from a mass of unintelligent words a clear, comprehensible system.

To accomplish the act of speaking correctly the brain must do two things: First, receive an impression, and second, express the idea. In other words, the function of the brain is that of an organ which first is being acted upon, and then, in response to the external stimulus, itself acts. To each function is given a special name. *Motor* is the name given to the function of expressing impressions; *sensory* is the name given to the function of receiving impressions. There are, therefore, two types of aphasia, *motor and sensory*. Motor aphasia means faulty expression. Sensory aphasia means faulty perception. Fix these two divisions clearly in mind. All other types of aphasia and all other words relating to aphasia may be classified under these two headings.

The next division of aphasia is a subdivision of these types, motor and sensory. To make this division intelligible, one must bear in mind that there are two main ways in which impressions may be conveyed to the brain, and that there are two ways in which the brain can express these impressions. These two chief ways of conveying an impression to the brain are by means of the two senses of seeing and hearing. The two ways in which the brain may express an impression are by the acts of speaking and writing. You will readily see, therefore, that there are two types of motor aphasia and two types of sensory aphasia. The important point at this stage is to have in mind what are these two kinds of motor aphasia and what are these two kinds of sensory aphasia. Inasmuch as the brain has two ways of receiving an impression, through hearing and seeing, an interference with either one of these two methods will cause sensory aphasia. The memories of words which are heard and of

words which are seen are stored up in certain parts of the brain, from which, later on, they can be drafted for use. These parts of the brain are called centers. In the case of words heard the center is called the *auditory word center;* in the case of words seen the center is called the *visual word center*. A lesion affecting either of these centers or a lesion affecting the fibers which connect these centers, either with themselves or with the two motor centers, will produce aphasia. The character of the aphasia will depend wholly upon whether the individual center is affected, whether the individual center plus the fiber leading to another center is affected, or whether more than one center is affected. The absolute location of these centers must be learned. The *auditory word center* lies at the upper surface of the left temporal lobe, in the near-lying posterior end of the first temporal convolution. The *visual word center* is situated in the angular gyrus.

The same principle holds true concerning the two motor centers. Here there are also centers in which the same process of storing-up is carried on. These, too, are called the centers of speaking and writing. They are situated in Broca's convolution, that is, the third left frontal, in the near-lying part of the precentral convolution. A patient, therefore, is suffering from *sensory aphasia* who hears, but not understandingly, and who sees, but not understandingly. That is to say, he may hear perfectly, but the sounds conveyed to him are unintelligible; the speaker appears to him to be talking in a foreign language. He sees, but the object as conveyed to him is not as it appears to the normal man. He expresses his ideas intelligibly. He cannot, however, converse,

nor answer written or spoken questions. A patient is suffering from *motor aphasia* who understands what is spoken and what he reads, but at the same time is unable to express his ideas in intelligible language, or to express them in intelligible written form. He is conscious of his defects; he is able to utter sounds. These may be the constant repetition of some particular word or small sentence, or they may be simply a jargon. The writing

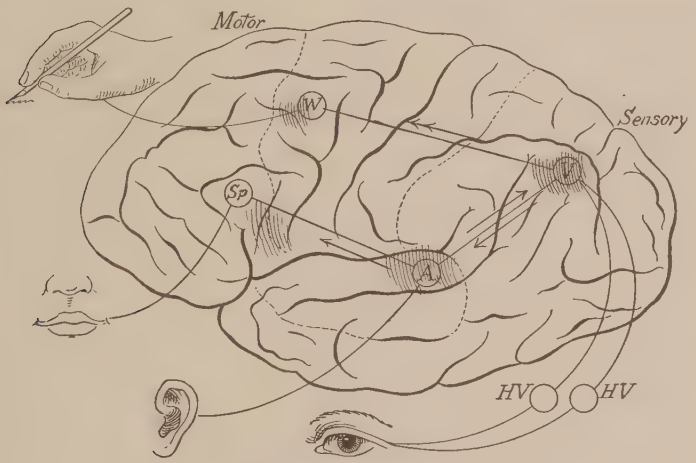


Fig. 55.—Parallelogram for examining an aphasic.

is of the same character. It may be one or two letters, parts of words, or simply strokes of the pen.

Causes.—The type of lesion which may cause an aphasia is usually circulatory, and, therefore, apoplectic. Rarely it is due to the presence of a new-growth, to abscess or softening. In the first instance it may be an embolus, a thrombus, or a hemorrhage. It is very difficult to differentiate among the three. Stewart, how-

ever, puts it quite aptly when he says: "Embolism produces the symptoms suddenly; hemorrhage takes several minutes; thrombus perhaps hours; abscess and tumors are still more gradual."

I have appended a diagram which is a combination of one from Bramwell and one from Stewart. Turn to it, and, with it before you, carefully follow these directions:

The base of the picture is an outline of the brain. There are four shadows on it—two in the fore part, two in the lower and posterior part. These four shadows represent the four centers; the two front ones are the centers for speaking and writing. They are the ones which are involved in motor aphasia. The two lower and posterior ones are the centers for seeing and hearing—the visual word center and the auditory word center. These are the two centers which are involved in sensory aphasia.

Draw a line connecting the two sensory centers with the two motor centers, that is to say, the visual word center with the writing, and the auditory word center with the speaking. Then connect the two sensory centers with each other, that is to say, the auditory word center with the visual word center. There is now practically a parallelogram. With this picture in mind, plan now the form of examination which you are going to follow.

EXAMINING AN APHASIC

In order to examine an aphasic, it is necessary to ask him questions which will prove or disprove the integrity of each center; secondly, questions which will establish the broken or unbroken connection from the one center to the other, and, thirdly, questions which

will prove or disprove the connection between three of those centers. Therefore, first ascertain if the center for speaking is intact. Then if the center for writing is intact. Then if the auditory word center is intact and the visual word center. Secondly, ascertain if the patient can receive an impression in either of the sensory centers and express it in the motor. Thirdly, if he can receive an impression in either sensory center, convey it to its fellow, and then transmit it to the corresponding motor center.

In examining an aphasic it is necessary to have two lists—one of articles and one of questions; also paper and pencil. The articles should be such as are in daily use, as: pencil, knife, scissors, coins, match-box, watch, keys, etc. The questions should be of the simplest and couched in plain language. The best are: "How are you?" "Where do you live?" "What day is this?"

The examination will take time and ought to be conducted in a systematic and regular way:

1. **Test for the Integrity of the Motor Speaking Center.**—In the examination of this center the point to ascertain is whether the patient can speak intelligibly. Note his ability to talk, to pronounce, his choice of words, and his method of talking spontaneously.

2. **Test for the Integrity of the Writing Center.**—The same questions should be asked and the same method of examination carried on in this as in the test of the speaking center, that is to say, have the patient write, and note the character of his writing, his method of writing, and his penmanship.

3. **Test for the Auditory Word Center.**—Here the questions should be directed to ascertaining whether he

comprehends what he hears. Direct him to perform certain simple acts, to point out the watch, the pencil, etc., and ask him questions the answers to which will convey information as to whether he comprehends what is being said.

4. **Test for the Visual Word Center.**—The point to ascertain in this test is whether the patient can comprehend written language. Write down a series of easy questions, such as the following, and ask the patient to answer them: "How old are you?" "Where do you live?" "What is your wife's name?"

5. **Test for the integrity of one sensory and one motor center, together with the fibers connecting them,** as, for instance, the visual center and the writing center. The object here is to ascertain the patient's ability to comprehend something which he sees, to store up that impression, and then to convey it in practical form. Have the patient copy a simple sentence, such as: "This is a fine day." "I have been very sick."

6. **The test for the integrity of the corresponding sensory and motor centers, together with the fibers connecting them,** namely, the auditory word center and the speaking. Ascertain if he can copy what he hears, if he can copy words and sentences which he has heard. Direct the patient to write from dictation simple sentences, such as, "This is a fine day"; also to repeat aloud after the examiner.

7. **Test for the integrity of the two sensory centers, together with one motor,** namely, the auditory word center, the visual word center, and the writing center. Can he comprehend what he hears, convey it to the visual word center, store it up, and then transmit it to the

writing center; in other words, can he write from dictation? Have the patient write a line from dictation.

8. **Test for the integrity of the two sensory centers, together with the other motor center,** that is to say, can he perceive something, transmit it to his hearing center, store it up, and express it through the speech center; in other words, can he read aloud, and by his hearing know he has read correctly? Have the patient read aloud.

In addition to these tests, finally he should be directed, without speaking to him, to imitate you. This is to establish his ability to perform gestures and pantomime movements. These two acts occur more or less invariably in aphasia.

These tests are quite sufficient to conduct a satisfactory examination of an aphasic. They represent, as you will see by looking at the diagram, every possible combination of centers, except putting together the two motor, which manifestly is impossible, and the very unusual routes of transmission from the auditory word center direct to the writing, and from the visual word center direct to the speaking. These are so unusual as not to be necessary for the student to concern himself.

Varieties of Aphasia

There are certain terms which you ought to know. These are:

Auditory aphasia.
Visual aphasia.
Motor aphasia.
Apraxia.
Alexia.
Agraphia.

Auditory aphasia is simply, as you have seen, beginning word-deafness. It may be of two types. This is dependent upon whether the lesion is cortical or subcortical. The latter type is exceedingly rare and need not concern you. In cortical word-deafness, or cortical auditory aphasia, the lesion is in the cortex itself; the patient not only suffers from word-deafness, but he has additional symptoms, due to the fact that the motor speech center is no longer controlled by the auditory center. The patient makes mistakes both in speaking and reading. He makes mistakes in spelling. He is able to talk, but his language is an absolute jargon because he is suffering from word-deafness and is unaware of his own errors. This is distinct from pure motor aphasia, where the patient recognizes his own mistakes as soon as he has uttered them.

Visual aphasia is another term for word-blindness, or **alexia.** Here the patient can see, but cannot comprehend. The letters are to him like those of a foreign language. Here, too, he may suffer from a cortical or subcortical lesion; the symptoms vary in accordance with it. In the subcortical type the incoming fibers are affected, though the visual word center itself remains intact. He knows what he wishes to write, and is able to write it, but once having accomplished this, he is unable to read it. In the cortical type, in addition to the symptoms described in the subcortical, the patient suffers from **agraphia.**

In **motor aphasia** the patient cannot express what he wishes in spoken words. Hughlings Jackson describes it very well when he says: "The patient is speechless, but not wordless." As a rule, he repeats certain phrases,

being at the same time conscious of his error. This variety is also either cortical or subcortical.

In treating the subject of aphasia, do not forget that I have described only the chief types, which are the distinct types. It is necessary to do this to render the subject intelligible and clear. Bear in mind always that the purely classic case is rarely the one which presents itself; that you are most apt to see not the pure type of aphasia, but combinations of these, irregular, unusual types, or even a complete destruction of all the centers, resulting in total aphasia. The cerebral artery is the one which supplies the speech centers. It is also the vessel which supplies the motor areas of the cortex and the corpus striatum; therefore, total aphasia and right hemiplegia often go together. Always bear in mind that the examination of a left-handed person must be exactly reversed, because in him the speech center is on the right. About nine-tenths of the people are right-handed, and it is therefore in very rare instances that this rule must be applied.

There is a form of aphasia which is not organic, and to which has been given the name of **hysteric aphasia.** It is fairly common, and one for which you must be on the lookout. Like most hysteric conditions, it is accompanied by other symptoms, including stigmata of degeneracy and peculiar psychic and motor symptoms.

Apraxia signifies the inability of a patient to perform certain regular, definite, muscular movements when he is suffering neither from physical nor mental impairment. Thus, a patient may be given a pencil, and may use it as he would a cigar, or when given a pair of scissors may use it as a button-hook.

The final division of aphasia is into **cortical** and **subcortical.** If the lesion is in any one of the centers, that is, the auditory, visual, speaking, or writing, the lesion is said to be **cortical.** If, on the other hand, the lesion is in the fibers running to the center, connecting the centers with each other, or leading from the centers, it is said to be subcortical. The symptoms of the two conditions will differ materially. It is evident that if the lesion is in the center itself, the damage will be greater, more extensive, and more pronounced.

It is, therefore, necessary to fix clearly in your mind the symptoms which occur in both the cortical and subcortical types of each one of these centers.

Symptoms of Motor Aphasia

Patient cannot express himself by spoken language. He is speechless, but not wordless (Hughlings Jackson).

(*a*) Symptoms of cortical motor aphasia:

Voluntary speech	Lost or impaired.
Repetition	
Reading aloud	
Voluntary writing	
Writing to dictation	
Copying	Retained.
Comprehension of spoken language	
Comprehension of written language	

(The latter impaired, according to Déjérine.)

(*b*) Subcortical motor aphasia:

Voluntary speech	Lost or impaired.
Repetition	
Reading aloud	
Reading	Retained.
Writing	
Comprehension of spoken language	

(*c*) Cortical sensory aphasia:

Voluntary speech conserved, but verbal paraphasia.

Copying retained	Lost or impaired.
Repetition	
Reading aloud	
Voluntary writing	
Writing to dictation	
Comprehension of spoken speech	
Comprehension of written speech	

(*d*) Subcortical sensory aphasia:

Voluntary speech	Retained.
Voluntary writing	
Copying	
Comprehension of written speech	
Comprehension of spoken speech	Lost or impaired.
Repetition	
Writing to dictation	

CHAPTER XVI

CEREBROSPINAL FLUID

The cerebrospinal fluid is a clear, limpid, colorless fluid, with a specific gravity of 1006 and an alkaline reaction. In taste it is salty, resembling tears and sweat rather than lymph and blood. It varies in amount from 100 to 130 c.c., while the pressure under which it flows fluctuates between 40 and 150 mm. Its main constituents are water and sodium chlorid. In addition, it contains glucose, albumin, carbonates, phosphates, urea, about 55 per cent. of CO_2, traces of globulin, the only protein material present, and a few cells. These cells are mononuclear, lymphocytic, and endothelial; in health their number varies from 2 to 10 per cubic centimeter.

The cerebrospinal fluid is neither a mere exudate nor a true secretion. It is derived from the plasma of the blood by dialysis pure and simple. Its main source of origin is the epithelial cells lining the choroid plexus of the ventricles; additional sources are probably the perivascular system of the nervous tissues and possibly also the arachnoid villi. It has a definite circulation of its own, passing from the choroid plexus into the lateral ventricles, and from them, by way of the foramen of Munro, to the mid-ventricular system. It then escapes by way of the foramina of Magendie and Luschka, and probably other apertures, which are not clearly understood, into the subarachnoid space. In the subarachnoid space it bathes both brain and cord, flowing from there

by way of the arachnoid villi or tufts, the dural sinuses, the veins, and possibly the lymphatics into the circulation. This idea is one generally, although not universally, accepted. Many maintain that no connection exists between the subarachnoid space of the cord and brain.

Cushing thinks that some of the cerebrospinal fluid may escape along the meningeal sheaths of the cerebral and spinal nerves, especially the sheath of the optic nerve. The secretion is active and continuous, so that when withdrawn it promptly re-forms, passing with each systole of the heart from the cranium to the spinal canal. It renews itself six or seven times in twenty-four hours, and consumes from twenty minutes to two hours to complete the circulation.

The **functions of the cerebrospinal fluid** are numerous. They may be classified as: (1) Mechanical; (2) chemical; (3) distributory; (4) interchangeable.

(1) The mechanical function is that of a water-bed and cushion. It serves to protect some parts of the nervous system from shock, to support others, and to aid in maintaining a uniform pressure.

(2) The chemical function is that of protecting the nervous system against infection. The choroid plexus, from which the cerebrospinal fluid is largely secreted, acts both as filter and ferment maker. It bars the entrance of certain deleterious substances, as has been proved by injections of colored fluids. It also creates antiferments which destroy and neutralize other ferments.

(3) The distributory function exercised by the cerebrospinal fluid is that of carrying to the tissues the active principles of the pituitary body.

(4) The interchangeable function is that of transferring to the blood, in its passage through the different spaces of the nervous system, carbon dioxid and water, and receiving in return from the blood oxygen and sugar.

Thus it will be seen that the functions of the cerebrospinal fluid are numerous and definite. Frazier aptly compares them to those of the lymph.

The **examination of the cerebrospinal fluid** is of great value in the diagnosis and treatment of certain forms of nervous diseases, especially of the different kinds of meningitis and syphilis. It should be made a routine part of the examination of every case of organic disease of the nervous system.

The distinct characteristics that should be sought for in this examination are:

(1) The naked eye appearance.
(2) The serology.
(3) The chemical examination.
(4) The cytological examination.
(5) The bacteriological examination.
(6) The pressure.

(1) **The Naked Eye Appearance.**—The fluid may be:

(*a*) Normal.
(*b*) Blood-stained.
(*c*) Yellow.
(*d*) Turbid.
(*e*) Purulent.

(*a*) The normal appearance of the fluid is clear and limpid. Fluid normal in appearance may still reveal pathological changes. Syphilitic fluids are almost invariably clear and limpid.

(*b*) Blood-stained fluid means one of two things, either

that the patient has sustained a hemorrhage or that the operator, in the course of the lumbar puncture, has pierced a small blood-vessel. To differentiate, draw the blood-stained fluid into three test-tubes. If all three, that is, the last as well as the first, are red, the stain is hemorrhagic and not due to the puncture. If the third or even the fourth vessel shows fluid which is yellowish, a hemorrhage has occurred. Hematoidin crystals will also be found.

(*c*) The spinal fluid is sometimes yellow. It is unusual, but does occur in jaundice and spinal cord tumor with compression. The shade of yellow is distinct and bright, not like that which follows hemorrhage. The explanation of its occurrence in compressive lesions of the spinal cord is as follows:

Compression of the cord forms a cul-de-sac more or less complete and distal to the site of compression. The changes of the fluid in this cul-de-sac are first an increase of protein material without an increase of cells, and later, as the compression persists and increases, a still greater increase of protein material with a change in the color of the fluid to an amber or yellow. After removal the fluid coagulates spontaneously, and may show an increase of cells, dependent on whether or not there is an involvement of the meninges. This syndrome, known as the "Nonne Froin," is diagnostic of a compressive lesion of the spinal cord.

(*d*) Turbid fluid is the usual type which accompanies acute meningitis, but not necessarily tubercular meningitis. It is of varying degree, from a slight, hardly perceptible cloudiness to a well-marked and distinct turbidity.

(*e*) Purulent fluid means either purulent meningitis or ruptured abscess. If the former, the particular type of micro-organism will reveal the particular type of meningitis.

(2) **The Serology.**—The Wassermann reaction is essentially a laboratory measure; it is not possible nor desirable that either student or general practitioner should undertake it. He ought to know and understand the principles involved. The Wassermann reaction is a measure or valuation of certain substances which are found in the sera and spinal fluids of syphilitics. It is a quantitative test. The report, therefore, should state definitely with what amounts a given specimen was negative or positive; it is not sufficient to say that the reaction was simply positive or negative.

(3) **The Chemical Examination.**—A. The chemical examination of the cerebrospinal fluid is important; it may show five different facts:

(*a*) The globulin content.

(*b*) The presence or absence of fibrin.

(*c*) The presence or absence of sugar.

(*d*) The presence or absence of cholin.

(*e*) The colloidal gold reaction. (See page 252.)

(*a*) Globulin is present in the normal fluid; conditions arise in which there is an excess of this albuminous content. These are tabes, general paresis, and cerebrospinal syphilis.

(*b*) Fibrin may be present in acute meningitis.

(*c*) Sugar is often found in the cerebrospinal fluid of diabetics, and sometimes in cases of brain tumor.

(*d*) Cholin sometimes occurs in cerebrospinal fluid. It is a product of myelin degeneration and is present in acute degenerative diseases of the nervous system.

B. The chemical examination of the cerebrospinal fluid ought to embrace three different examinations:

(*a*) The tests for an increased globulin content.

(*b*) Reduction tests.

(*c*) The colloidal gold test.

(*a*) **The Tests for an Increased Globulin Content.**—A positive globulin test means the existence of an organic disease of the central nervous system. It does not necessarily mean the presence of a syphilitic disease.

There are three different tests which may be used to ascertain the globulin content. These are:

(1) The Ross-Jones modification of Nonne's Phase I reaction.

(2) Pandy's test.

(3) Noguchi's butyric test.

(1) The Ross-Jones modification is simple, satisfactory, and delicate. Place 1 c.c. of the spinal fluid in a small test-tube. Introduce at the bottom of the test-tube, by means of a small glass pipette, an equal amount of a neutral saturated solution of ammonium sulphate crystals, which has been brought to saturation at the boiling-point. If the globulin content is increased there will form at the point of contact of the two fluids a grayish-white ring. If the fluid is normal no ring will form.

(2) Pandy's test is the simplest and quickest of the three. Place 1 c.c. of a saturated aqueous solution of carbolic acid in a small test-tube. Add 1 drop of the spinal fluid. If the globulin content is increased there will form at the point of contact of the two fluids a ring bluish-white or cloud-like in character. If the fluid is normal no ring will form, or, at most, an extremely faint opalescence.

(3) Noguchi's butyric acid test has two great disadvantages—it requires two hours and gives forth an insufferable odor.

(*b*) The **reduction tests** are of comparatively little importance. All normal fluids give a faint reduction of Fehling's solution. It is said to occur in 100 per cent. of the cases of general paresis.

(*c*) The **colloidal gold test** is rapidly assuming a place of the first importance in the study of syphilis of the nervous system. It ranks with the Wassermann reaction and the cell count, and should be made a routine part of the examination of every suspected case of nervous syphilis.

Szigmondy showed that a solution of colloidal gold when brought into contact with certain albuminous bodies in the presence of an electrolyte, such as sodium chlorid, would cause a clumping together of the small colloidal particles, producing various changes in color and even precipitations. He also noted that various albumins reacted differently in these respects—*i. e.*, the point at which precipitation and color change would take place. Carl Lange applied this method to the study of the spinal fluid, and while observing the reaction of the colloidal gold chlorid solution of the spinal fluid in a case of general paresis, observed that a certain color change took place, which when repeated with the spinal fluid of other cases of paresis, gave the same characteristic results; and also that other color changes occurred in other spinal cord conditions. Thus he was enabled to find at least five different shades.

To simplify and facilitate the charting, Lange applied to each shade a numeral. The normal red shade which

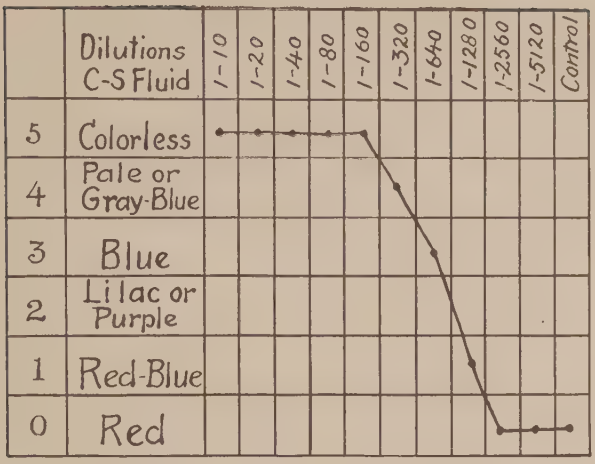


Fig. 56.—Curve typical of general paresis.

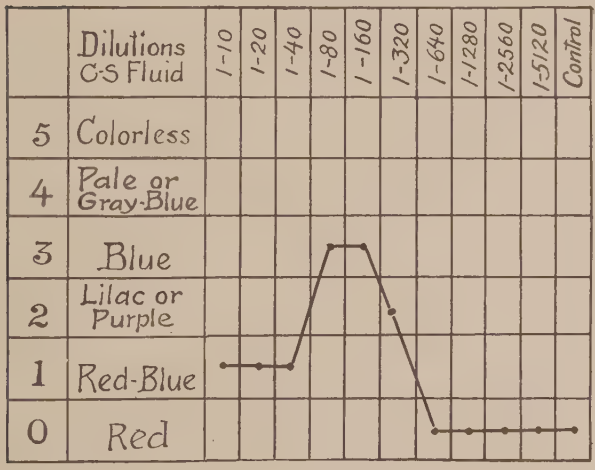


Fig. 57.—Luetic curve.

is the color reaction of every normal spinal fluid to the reagent became zero; the next, the red-blue, one; the lilac,

two; the blue, three; the pale or gray-blue, four; and the colorless, or the one in which occurred the maximum amount of precipitation, five. He then devised a chart, assigning to each test-tube a column and to each color change a number. He placed a dot in the column assigned to each test-tube, and then connected the dots, as is done in a fever chart. In this way he constructed a chart which gave definite curves. He found that most

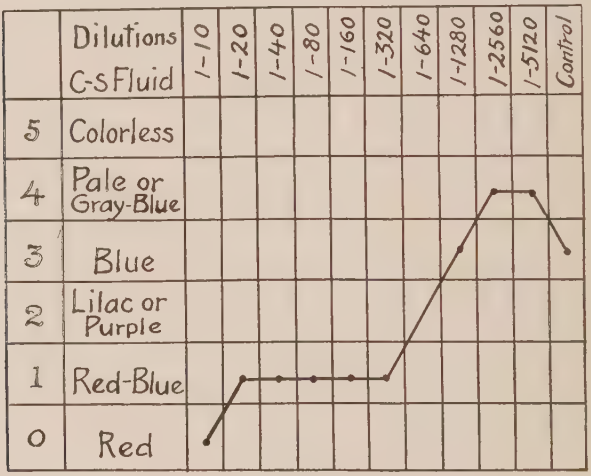


Fig. 58.—Meningitic curve.

cases of general paresis gave the same curve, and that most cases of cerebrospinal syphilis gave a particular curve, and that the meningitic cases gave a definite curve. There are, therefore, three distinct and definite curves—the paretic, the luetic, and the meningitic.

To these color changes due to alterations in the dispersion value of the colloidal gold are given the name reaction. The reaction which results in no color change is called negative, and is the one characteristic of normal

spinal fluid; the reaction which results in complete decolorization is called markedly positive, and is the one characteristic of general paresis.

The importance of this test lies more in its prognostic than diagnostic value. By this I mean that it is possible to take a patient who has the clinical signs of syphilis and state definitely from the colloidal gold test what definite form his syphilis will take in the future. The test, therefore, becomes one of great value as an indicator of the future treatment. A case which has been clinically diagnosed as cerebrospinal syphilis might show a characteristic paretic curve following a colloidal gold test. Again, a case which clinically would show symptoms of general paresis might, in the colloidal gold test, illustrate simply a luetic curve.

(4) **The Cytological Examination.**—The cytological examination gives the most important information of all the different tests. It is of especial value in meningitic and syphilitic cases both from a diagnostic and therapeutic point of view, and it is therefore of paramount importance that the student familiarize himself with the cell count.

There are three observations to make in the cytological examination:

(*a*) The number of leukocytes present in the field.

(*b*) The types of cells present, together with their relative proportions.

(*c*) The presence of other elements.

(*a*) To count the cells a counting chamber has been devised. This consists of a square of glass ruled in such a manner as to allow the examiner to count the cells lying in a given space. It is an advantage to use the Fuch

counting chamber, inasmuch as it is $\frac{2}{10}$ high. The number of cells present in the normal field varies; it should not exceed 10 per cubic millimeter of fluid; above 10 is pathological. An increase of cells is the result of an active inflammation. The cells may vary from 10 to 100, or even up to several thousand, according to the virulence of the infection, the particular strain of spirochetæ, and the type of the lesion.

The following is a list of the diseases in which occurs an increase of cells:

1. Cerebrospinal meningitis.
2. Tuberculous meningitis.
3. General paresis.
4. Tabes.
5. Cerebrospinal syphilis.
6. Taboparesis.
7. Secondary syphilis not affecting the nervous system.
8. Poliomyelitis.
9. Trypanosomiasis.
10. Mumps.
11. Herpes zoster.
12. Brain tumor.
13. Chronic alcoholism.

(*b*) The type of cells present varies. There may be mononuclear and polynuclear leukocytes.

Lymphocytosis indicates a chronic meningeal irritation. It is especially pronounced in general paresis, tabes, and cerebrospinal syphilis, and particularly in the earliest stages. Intraspinous treatment frequently reduces the lymphocytosis to normal because it reduces the irritation.

Leukocytosis is the ordinary accompaniment of epi-

demic meningitis, pachy- lepto-, and acute cerebrospinal meningitis. In these diseases the increase is largely due

Fig. 59.—(Copied from Plaut, Schottmüller, and Rehm.)

to polymorphonuclear cells. In tuberculous meningitis the leukocytosis is made up of mononuclear cells.

(*c*) The presence of other elements. These may be fibrin, blood-cells, epithelial cells, detritus, nerve-tissue, and micro-organisms.

(5) The **bacteriological examination** shows the presence of micro-organisms. In performing this examination it is of considerable advantage to culture the spinal fluid when withdrawing it. There are many types of micro-organisms found in the spinal fluid. It is not necessary for the student to be familiar with all, but only the most common ones. These are: the meningococcus, the tubercle bacillus, the pneumococcus, and the streptococcus. In the study of the meningococcus a distinction should be made between the acute and secondary forms, the latter being the infectious type.

(6) The **pressure** may be normal, increased, or decreased. Normally, following a lumbar puncture the fluid should flow drop by drop, that is to say, one drop every two or three seconds; a decrease of pressure will be evident by a greater interval between the drops; an increase by a steady flow of the fluid or even a gush. Instruments have been devised to register more accurately the degree of pressure. Frazier recommends a small mercury manometer which can be attached to the lumbar puncture needle in such a manner as to register this degree of pressure even before any of the fluid has been withdrawn. Quincke states that the normal average pressure is between 40 and 60 mm. water. There is no standard. Authorities differ greatly, some putting the normal pressure as high as 150 mm. Above this latter figure it certainly becomes pathological. High cerebrospinal fluid pressure is of far greater importance than low; it is significant of organic disease of the nervous system.

Quincke states that a moderate increase of pressure with severe pressure symptoms points to an acute process, whereas a great rise of pressure with slight pressure symptoms points to a chronic process.

The increase of pressure is present in many of the cerebral conditions, such as tumor and meningitis; it is not, however, limited to these, but is found also in certain febrile diseases, for instance, pneumonia and influenza. In epidemic cerebrospinal meningitis an increase of pressure is constantly present, practically throughout the disease.

The only change which occurs in quantity is an increase. This may be brought about in one of three ways: (*a*) inflammatory lesions; (*b*) decrease of blood-pressure; (*c*) venous obstructions.

Lumbar puncture is the name given to the procedure employed to obtain cerebrospinal fluid. It may be done for diagnosis, for treatment, or for prognosis.

As a diagnostic aid it is of great value in nearly all organic nervous diseases; it is especially of value in those diseases resulting from syphilis. It is of value for treatment in those cases where there is an oversecretion of fluid, where the fluid is under great pressure, and where it is advisable to introduce sera or other fluids into the spinal canal. It is of value in prognosis in syphilitic cases where certain tests, such as the colloidal gold, render possible an opinion as to the future course of the disease.

Lumbar puncture is now being accorded a position of importance in every examination of a nervous patient; it should be a routine part of such examinations. So common is syphilis and so usual is it to find a negative Wassermann test in the blood in syphilitic cases that

a lumbar puncture should be made in the examination of every nervous condition. Every patient with acknowledged syphilis should be punctured, and every patient with suspected syphilis should be punctured. As I have said, it is a very frequent occurrence to find a patient with a negative Wassermann report on the blood who will yet show a positive Wassermann report on the spinal fluid. Therefore, the absence of a positive Wassermann test in the blood is not an indication to defer a lumbar puncture, but rather is it an indication to hasten one.

Lumbar puncture is a simple and practically painless operation of a very minor degree. The patient rarely suffers any pain during the insertion of the needle, and with proper precautions and care the after-results should be very slight.

The student should realize the importance of the test, and the patient should be educated to the importance of the additional information afforded by a lumbar puncture.

There are two ways of performing the lumbar puncture: one with the patient sitting up, the other with the patient lying down. The method of sitting up is the better, more comfortable for the patient, easier for the operator, and one which is more liable to give good results. The objection to having the patient lie down is that the average bed is either too high or too low, and that the patient cannot get near enough to the edge, as the spring or mattress is apt to yield and sag. There are no objections to puncturing a patient in the upright position, but there is a marked objection to the patient remaining in that position after the puncture. The precautions to take,

therefore, are twofold: one, to have the patient ready and prepared to go to bed immediately after the puncture and to remain there for twenty-four hours; the other, to have the patient drink a great deal of water in the two or three hours preceding the puncture. This precaution is taken in order to assist nature in restoring the loss of fluid which the puncture entails. It will also decrease the subsequent headache. The position of the patient during the puncture in no way affects the subsequent headache or nausea.

The best way to perform a lumbar puncture, therefore, is to have the patient sitting astride a small, straight-back chair, so that he will face the back of the chair and lean forward, resting on his arms, which in turn are resting on the back of the chair. In this way the back of the patient is directly in front of the operator and is at the same time bowed, so as to cause the spinal column to assume a convex shape. The importance of this lies in the fact that it stretches and therefore enlarges the space between the vertebræ, thus giving the needle greater leeway. The site selected for the puncture should be the space either between the fourth and fifth lumbar vertebræ or that between the third and fourth. These two are the widest, and of the two the space between the fourth and fifth is slightly the larger.

It is very easy to find these spaces. Draw a horizontal line across the patient's back at the level of the iliac crests. This line intersects the vertebral column at the tip of the fourth lumbar spine. The puncture should be made immediately below this. The best needle to use is one made of platinum, as it will not rust and the canula is much less apt to become plugged. The needle should

be of sufficient length to go through several tissues which are met before the vertebral canal is reached—10 centimeters or just over 4 inches is the best length for a needle. The strictest antiseptic precautions should be used. The needle should be boiled and the skin of the patient painted with iodin.

The operator, with hands carefully washed and sterilized, should then feel with the left index-finger for the vertebral space selected, making a dent with the finger in the spot where the needle is to be inserted. The operator, with hands carefully washed and sterilized, should then feel with the left index-finger for the vertebral space selected, making a dent with the finger in the spot where the needle is to be inserted. When this has been done he should hold the needle hilt between the first two fingers of the right hand, and, using the left forefinger as a guide, push the needle inward and upward toward the patient's umbilicus. Care should be exercised to have the needle in the subarachnoid space. The best indication that the needle is in this space is the sensation of freedom, together with the ability to move it back and forth. When the operator is satisfied that the needle is in the proper place, he should withdraw it, leaving *in situ* the canula. The fluid should then flow drop by drop. If no fluid is obtained, the operator should make a second attempt. A dry tap is no tap at all, and in the vast majority of cases is the fault of the operator. The drops of the fluid ought to fall into two test-tubes, part in one, part in the other. Never draw off more than 10 c.c. The patient should then return to bed and remain for at least twenty-four hours in a recumbent position. This precaution is taken to avoid

violent head- and back-aches, which are inevitable if the patient rise too soon. It is imperative to restore the fluid at as early a date as possible; the way to restore it being by rest. The headaches result from a disturbance of cerebral pressure.

Dangers of Lumbar Puncture.—The bad results which may occur from lumbar puncture are three: The needle may not enter the spinal canal; it may go beyond the spinal canal into the membrane beyond or it may become clogged with blood. In the first contingency, by pushing the needle a little further in, the canal may be reached and the difficulty averted; in the second case, the reverse measure of drawing the needle a little bit out may place it in the spinal canal, and in the third case the difficulty of clogging the needle may be averted by removing and once more inserting the canula. The greatest danger which may result from lumbar puncture is sudden death from pressure on the medullary centers. This may occur in tumor of the brain and in those conditions in which the intraventricular pressure is excessive. Here sudden withdrawal of the fluid may result in sudden death.

It has been proved that it is possible to have the needle inserted in the spinal canal and yet not draw forth any fluid. Dr. W. S. Leszynsky placed the needle in the fourth lumbar space and obtained a dry tap, he then inserted a needle and canula in the lumbar space just above, injecting a sterile salt solution into this, which in turn came out through the original puncture, thus proving that the original needle was in position.

Diseases in Which the Spinal Fluid is Altered.—The diseases which are altered by the cerebrospinal fluid may be divided into two large classes:

(*a*) Those which are syphilitic.

(*b*) Those which are non-syphilitic.

(*a*) In **syphilitic diseases** the cerebrospinal fluid is changed in both its physical and chemical properties, and in most it is changed in respect to the cell count. It is not changed in that there are present any micro-organisms. This latter is the main difference between syphilitic and non-syphilitic diseases. In the syphilitic diseases there is almost always, as I have said, an increase in lymphocytes. The amount of this increase is entirely dependent upon the degree of involvement on the part of the spirochetes of the nervous system.

It should not be forgotten that in some cases of syphilis, especially in general paresis and in a few cases of cerebrospinal syphilis, the cell count toward the end of the disease drops so as to become almost normal. The list of syphilitic diseases in which the cerebrospinal fluid is altered are:

1. General paresis.
2. Locomotor ataxia.
3. Cerebrospinal syphilis.
4. Taboparesis.

1. *General Paresis.*—The examination of the spinal fluid in general paresis shows a positive Wassermann in the fluid even in very low amounts, an increased number of cells, and an increased amount of globulin. The increase of the number of cells is usually not very great, not exceeding 25, and averages in the neighborhood of 18 to 22. It is in the more acute forms, the cerebrospinal forms of syphilis, in which there is an active inflammation that we find the count very greatly increased.

2. *Locomotor Ataxia.*—The examination of the spinal

fluid in locomotor ataxia shows various results. Kaplan has classified the cerebrospinal fluid of tabes under four different headings. I do not think, however, that it is necessary for the student to burden himself with all these differential examinations. The important form is the examination of the serology of the usual type of tabes. In this the Wassermann reaction in both blood and fluid is more apt to be positive than negative. In a few cases it has proved negative. The globulin is plus or minus, usually plus. There is always a prompt reduction of Fehling's test, and there is a large increase in the number of cells, far greater than in cases of general paresis. The usual range of cells is from 20 to 100, and there are over 95 per cent. of lymphocytes. The variation of the cell count is, of course, dependent upon the activity of the disease.

3. *Cerebrospinal Syphilis.*—The examination of the spinal fluid in cerebrospinal syphilis depends on whether the condition is purely cerebral or purely spinal. The spinal fluid is positive in at least three-fourths of these cases, the excess of globulin is considerable, being over 25 per cent., and usually the Fehling reduction is present. The increase in cells is a very constant and prominent sign in cerebrospinal syphilis, and varies according to the particular pathological lesion, being increased, as I have said above, in the meningitic types. The cell count may vary from 30 to 40 cells to 500, or even more.

4. *Taboparesis.*—The examination of the spinal fluid in taboparesis gives a positive Wassermann. It gives a reaction of 90 per cent. when using a small amount of the fluid, and 98 per cent. when large amounts are used. There is an increase in the number of cells in almost

every case, the number, however, not being particularly great, usually from 20 to 30. There is usually an increase in globulin.

(*b*) In the **non-syphilitic diseases** the cerebrospinal fluid is altered in several ways—physically, chemically, in the number of cells, in pressure, and in the presence of micro-organisms.

The physical alteration is in the color and consistency of the fluid, which, instead of being clear and white, becomes thick, gray, and purulent. The specific gravity rises, and if the fluid is very purulent reaches 10, 20, or 30. The chemical alteration consists of an increase of the globulin, together with an addition of several protein bodies. The cells in all the non-syphilitic diseases, and especially in those of the meningitic type, are greatly increased in number. The change in pressure is one of increase, which is especially marked in the meningitic diseases. The most important change of all in the spinal fluid of non-syphilitic diseases occurs in the bacteriology. Most of these non-syphilitic diseases are due to a particular micro-organism. This organism circulates in the spinal fluid and is demonstrable under the microscope. This, therefore, is the important change. There are many of these micro-organisms which are easily demonstrable and many others which are questionable. The most frequent and most common type are, of course, the meningitic group.

In the *epidemic cerebrospinal type of meningitis* there is the specific organism known as Weichselbaum's diplococcus. In *influenza meningitis* there is the organism described by Pfeiffer. In the *gonococcic meningitis* there is the particular micro-organism of Neisel. In the *tuber-*

culous meningitis the tubercle bacillus may be found. In the *pneumococcic meningitis* there is present the pneumococcic bacillus. In the *staphylococcus meningitis* there will be found the staphylococcus organisms. In the *streptococcic meningitis* there will be found the streptococcic organisms. In addition to these micro-organisms, which all authors agree may be found in the spinal fluid, there are many other bacteria in which there is some question of doubt as to their presence. Several authors have reported to have discovered them in the spinal fluid, while others have questioned their presence. It will be well for you to have simply a list of those which have been reported:

1. Bacillus coli communis.
2. Bacillus pyocyaneus.
3. Bacillus mallei.
4. Bacillus anthracis.
5. The saccharomyces.
6. The actinomyces.

In addition to these two types, the syphilitic and the meningitic, there are few conditions in which the examination of the spinal fluid becomes of any great value. It is only in those instances in which the syphilitic infection complicates the disease that the examination of the spinal fluid proves of any value. In the various types of neuritis, in epilepsy, in functional conditions, and in the organic psychoses the lumbar puncture gives no new information.

Conditions in Which the Spinal Fluid May Serve of Value in Treatment.—This can again be classified under the two headings of syphilitic and meningitic. In the former the spinal fluid may be used as a vehicle in which

to inject certain drugs which have a property of destroying the spirochete, such as salvarsan and bichlorid of mercury. In the latter it may be used as a vehicle for certain serums which have a neutralizing tendency on the specific organism of meningitis. In these cases it is best to substitute an equal amount of salt solution. Instances occur in which it may become necessary to draw off a certain amount of spinal fluid for the purpose of reducing pressure. There are conditions in which the removal of a certain amount of spinal fluid is a benefit. In *alcoholic* conditions, in which delirium tremens is present, it has been found that a removal of small amounts of the spinal fluid cause a very marked and beneficial effect upon the delirium. In *hydrocephalus* and *poliomyelitis* the withdrawal of the fluid is beneficial in reducing pressure.

CHAPTER XVII

ELECTRIC REACTIONS

In health electric stimulation of a muscle causes a contraction which follows certain definite laws and affects the different poles in a definite manner. The character of this contraction is sharp and jerky. The polar reaction is considerable, but unequal, and the response made to the negative pole is greater than that made to the positive.

To examine a patient electrically requires either a portable or a wall battery. It must supply both the galvanic and faradic currents. Two connecting wires and two sponge electrodes are indispensable. One electrode must have an interrupter, that is, a key which will make it possible to make or break the current. In this way it is possible to interrupt the current and so study the muscular contractions.

Immerse the sponges in warm water to which has been added a little salt. Warm water is more agreeable to the patient, and the saltiness causes a stronger reaction. Before applying, squeeze the sponge electrodes, to remove the surplus water and avoid wetting the patient.

Place one electrode on some definite, fixed part of the patient, preferably the sternum or the back of the neck. Place the other electrode on the part to be examined. This latter should be the smaller of the two, so as to make possible the selection of particular muscles and motor points. On this electrode place the interrupter.

It is necessary to place the smaller, or examining electrode over certain fixed points of the muscles. This is essential, because at these points the stimulation of the current is at its maximum, and, therefore, a stronger, more obvious contraction results. These points are called the motor points. They can be learned only by

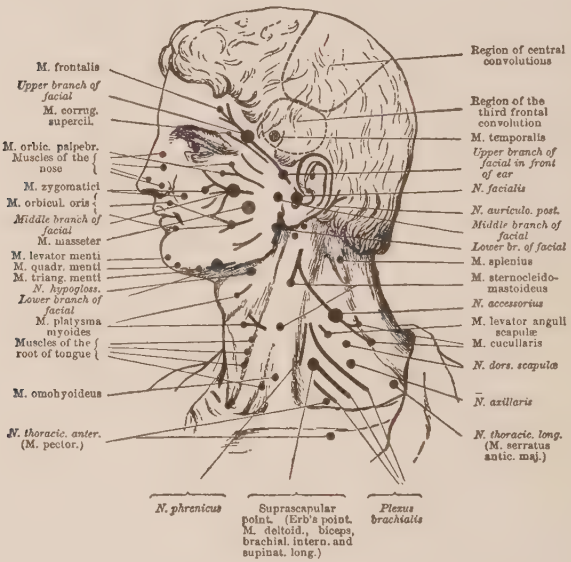


Fig. 60.—Nerves and motor points in face and neck (Erb).

reference to the tables on the following pages. They are called motor points because they are the points at which the motor nerves emerge from the muscle or those at which the nerves are nearest the skin. Therefore, being nearest the electrode, stimulation at these points gives the best results.

Place the limb or part to be examined so as to afford

the best results. In the case of the hand, flex the fingers and avoid any active resistance. In the case of the arm avoid rigidity and let it hang loosely.

In examining with the galvanic current, if it is difficult and troublesome to decide which pole is positive and

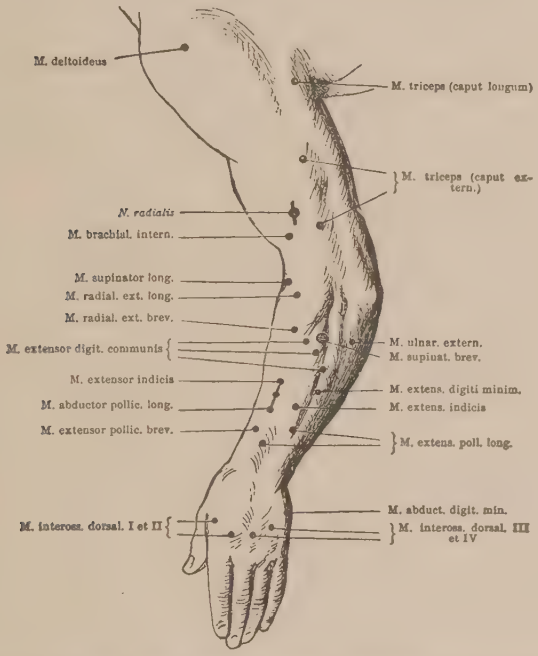


Fig. 61.—Nerves and motor points in upper extremity (Erb).

which negative ascertain definitely in this way: remove the electrodes, put the ends of the wires in a glass of water, turn on the galvanic current. A steady column of bubbles will rise from the negative pole.

Always test the muscle of the healthy side first, as

this will enable one to establish a standard. Always test each current on yourself before applying it to the patient, thus avoiding a shock to him. Always begin with a current weaker than is necessary, gradually increasing to a point where the required strength is reached. This should be sufficient to bring about a strong contraction. It is necessary to have connected with the instrument a galvanometer, which will measure the amount of current used.

It is the nerve which responds to faradic current, and, although the muscle responds by a contraction, it in all probability receives the stimulus only through the nerve. The galvanic current causes a contraction through direct stimulation of the muscles themselves, the nerve exercising only a controlling influence.

KCC>ACC>AOC>KOC

When there is a lesion in the lower motor neuron the order of susceptibility becomes ACC>KCC>KOC>AOC.

In practical testing it is the closing contraction on which the attention is usually centered, for, if ACC is greater than KCC, that in conjunction with the other evidence is sufficient to indicate abnormal reaction without troubling about the opening contractions.

Practical Considerations.—1. If at first too strong a current is used, the resulting contraction is too widespread to give any reliable information. In the general effect the reaction of any particular muscle or group of muscles is lost. The unpleasant sensation causes the patient to lose confidence, making further testing difficult, beside causing pain to the patient.

2. The farther away the muscle under observation is from the central axis of the body, the greater, even under

normal conditions, is the strength of the current required to produce a contraction.

3. The resistance of the skin to the passage of the electric current is very great, and time, patience, and constant wetting of the skin are necessary to overcome the resistance.

4. In fat people patience is required to give the electricity time to get through the skin and fat to the muscle.

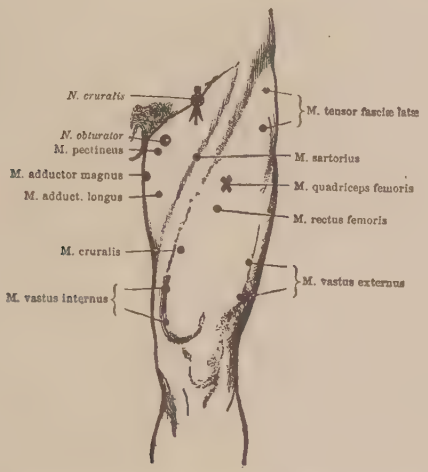


Fig. 62.—Nerves and motor points in lower extremity (Erb).

Careful differentiation is sometimes necessary to distinguish between a normal fat limb and a pseudohypertrophic dystrophy.

Abnormal Reactions.—In addition to the reactions which I have already described, there occur in medicine, and especially in neurology, several reactions which are unusual and, therefore, abnormal. I have classified these under six headings. The first comprises the increase of

both the faradic and the galvanic currents. The second heading is the reverse of the former, and comprises those conditions in which there is either a diminution or an absence of both the faradic and galvanic currents.

A list of these follows:

A. Increased to both faradic and galvanic currents:

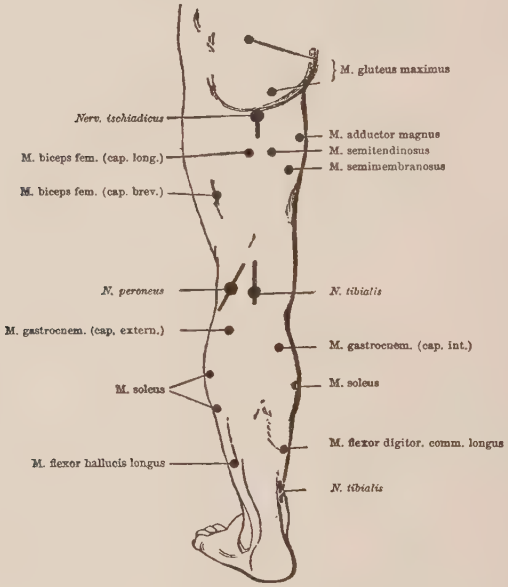


Fig. 63.—Nerves and motor points in lower extremity (Erb).

1. Undue irritability, such as in chorea and early spastic paralysis.

2. Diseases characterized by profuse sweating (which diminishes the resistance of the skin to the electric current), as in Graves' disease.

3. Hysteria, in which there is sometimes an increased susceptibility to faradism.

B. The reaction of degeneration (R. D.) indicates, as a rule, a lesion of the lower motor neuron type; in the upper motor neuron type there is no such reaction.

The following table gives the diseases in which the reaction of degeneration occurs:

1. Diseases of the anterior horns:

(a) Poliomyelitis.

(b) Amyotrophic lateral sclerosis.

(c) Spinal gliosis.

(d) Diffuse cervical and lumbosacral myelitis (with involvement of the gray matter).

2. Diseases of the nuclei of the bulbar nerves corresponding to those of the anterior horns (progressive bulbar paralysis, acute inferior polioencephalitis, etc.).

3. Diseases of the anterior roots:

(a) Compression from tumors and thickening of the meninges, as in syphilis, cervical hypertrophic pachymeningitis.

(b) Compression in the intervertebral foramina in diseases of the spinal column (caries, tumor, fracture, dislocation).

4. Severe affections of the peripheral nerves:

(a) Traumatic (section, severe bruising, pressure by tumor).

(b) Rheumatic (facial paralysis).

(c) Toxic and infectious:

Lead paralysis.

Alcoholic paralysis.

Arsenical paralysis.

Infective forms of multiple neuritis, etc.

In addition to those two headings there occur three other abnormal reactions. These are called (C) myas-

thenic reaction, (D) tetanic reaction, and (E) myotonic reaction. Each reaction has characteristics of its own, and each is diagnostic of the particular condition in which it occurs. Each will, therefore, merit a short description, and the student must know each.

The myasthenic reaction occurs in the condition known as myasthenia gravis. The main characteristic of this abnormal condition consists of the gradual exhaustion

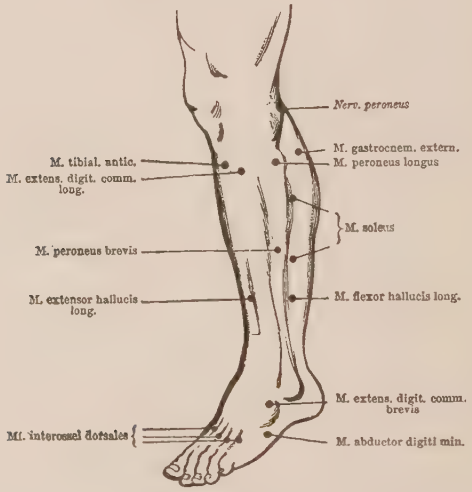


Fig. 64.—Nerves and motor points in lower extremity (Erb).

of the muscular responses. There is no reaction of degeneration. There is neither irritability nor marked susceptibility. There simply occurs, following rapid stimulation, a gradual exhaustion of the muscular responses, both of the faradic and galvanic currents. The second characteristic of this reaction is that, after the tired muscles are given a short period of rest, they again respond to both currents. The myasthenic reaction,

therefore, is one in which the response of the muscles to both the electric currents becomes weaker and weaker until finally it ceases.

The myotonic reaction occurs in myotonia, or Thomsen's disease. Its main characteristic is an abnormal susceptibility on the part of all the muscles to the electric current. The response of the two poles is about equal, but all responses are prolonged and tonic. The muscles display an unusual degree of mechanical excitability, even to blows, especially, however, to the electric stimulation.

The myotonic reaction is, in some respects, a counterpart of the myasthenic.

The tetanic reaction occurs in tetanus. In this reaction there is a marked increase of the electric excitability of the sensory as well as of the motor nerves. The increased excitability of the motor nerves is far more constant than that of the sensory. In fact, it is found in all instances, even in those in which the current is weak.

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